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ДРУЖБЫ НАРОДОВ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»**

**КАФЕДРА ТЕРАПЕВТИЧЕСКОЙ СТОМАТОЛОГИИ С КУРСОМ ФПК И ПК**



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**Терапевтическая стоматология  
для студентов 3 курса**

**Therapeutic Dentistry for the 3<sup>rd</sup> year student**

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## **PREFACE**

This educational and methodical edition is based on the Educational program of the higher educational establishment on academic discipline for 1-79 01 07 “Stomatology” specialty. It is intended for 3rd year students of the Stomatological Faculty studying therapeutic dentistry in English.

It contains two sections - 5th semester and 6th semester.

The 5th semester contains educational material for 19 lessons. This section discusses the issues of antiseptics and sterilization in dentistry, individual and professional hygiene, cariesology, non-carious lesions. Materials for this part were previously published in «Guideline on therapeutic dentistry for the 5-th term» (2016). In the new edition, some of the topics have been revised and supplemented. Added new topics, such as «Methods of investigation characterized destructive process of periodontal tissues», «Possibilities of filling material choice in different cases», «Minimal invasive methods of operative treatment of dental caries».

In the 6th semester, theoretical aspects of pulp and periodontal disease, some issues of materials science, the main stages of endodontic treatment are covered in detail. This section presents material for 16 lessons.

The edition complements the existing educational literature on therapeutic dentistry.

## **5 SEMESTER**

### **LESSON 1.DEONTOLOGY IN THERAPEUTIC DENTISTRY. DENTAL OFFICE. STERILIZATION OF DENTAL INSTRUMENTS. THE BASIC RULES OF ERGONOMICS IN THE WORK OF THE DENTIST**

The questions to be studied for the learning of the topic:

1. Therapeutic dentistry as a science, subject matter, objectives, goals.
2. Structure and equipment of dental office. The basic requirements are presented to him.
3. Aseptic and antiseptic for dental seeing, definitely, main types.
4. Sterilization dental instruments, concepts. Main types of sterilization used in clinic therapeutic dentistry.
5. Key terms in the ergonomics dentists. Workplace of dentist.

#### **Question 1. Therapeutic dentistry as a science, subject matter, objectives, goals.**

Therapeutic dentistry is the science that studies the characteristics of diagnosis, clinics, treatment, prevention and rehabilitation of patients with diseases of hard tissues of teeth, periodontal, oral mucosa.

*The goal of therapeutic dentistry:* implementation of effective prevention, diagnosis and conservative treatment of diseases of the teeth, periodontal and oral mucosa.

*Objectives of therapeutic dentistry:*

1. Prevention and treatment of caries and its complications.
2. Prevention and treatment of non-carious lesions.
3. Treatment and prevention of periodontal disease.
4. Prevention and treatment of oral mucosa.
5. Clinical examination of dental patients.

The term "deontology" comes from the Greek words: «deontos» is due, proper and «logos» - knowledge, means the totality of ethical standards of professional conduct for health workers.

Ethics is defined as a branch of philosophy that deals with thinking about morality, moral problems and moral judgments.

**Basic ethical principles in dentistry.** They most certainly, coincide with those in medical practice and other health care disciplines which are described as universal ethical principles. Among them are classic ethical principles that are the same since Hippocrates age. During the previous two decades, some new ethical principles have evolved in doctor-patient relationship. Dentist has to be familiar with both classic and modern moral principles, to respect and implement them unequivocally. Principles of ethics

for the dental profession are that “the dentist should act in a manner which will enhance the prestige and reputation of the profession”. The principles of ethics are the aspiration goals of the profession. They provide guidance and offer justification for the code of professional conduct and the advisory opinions. For their significance some of the ethical principles are predominating. These are: avoiding of making any damage to patient; doing well for patient; autonomy of patient; patient’s informing. These principles are supplemented by other ethical rules like: altruism, justice, confidentiality, loyalty, truthfulness and other. All noted principles are overlapping in meaning and resulting one from another.

### **Principles of ethics**

The principles of ethics are as follows:

**1. Patient autonomy (self-governance).** It is based on the principle of respect for persons. Independent actions and choices of an individual should not be constrained by others and they should be respected. The dentist has a duty to respect the patient’s rights to self-determination and confidentiality. Professionals have a duty to treat the patient according to the patient’s needs, within the limits of accepted treatment, and to protect the patient’s confidentiality. The dentist’s primary obligations include involving patients in treatment decisions in a meaningful way, with due consideration being given to the patient’s needs, desires and abilities, and safeguarding the patient’s privacy.

**2. Nonmaleficence (do no harm).** The dentist has a duty to refrain from harming the patient. Professionals have a duty to protect the patient from harm. The dentist’s primary obligations include keeping knowledge and skills current, knowing one’s own limitations and when to refer to a specialist or other professional, and knowing when and under what circumstances delegation of patient care to auxiliaries is appropriate.

**3. Beneficence (do good).** The dentist has a duty to promote the patient’s welfare. This principle expresses the concept that professionals have a duty to act for the benefit of others and the dentist’s primary obligation is service to the patient and the public-at-large. The most important aspect of this obligation is the competent and timely delivery of dental care within the bounds of clinical circumstances presented by the patient, with due consideration being given to the needs, desires and values of the patient.

**4. Justice (fairness).** The dentist has a duty to treat people fairly. Principle of truthfulness comprises dentist’s sincerity toward patients, truth telling, never deceiving. This principle expresses the concept that professionals have a duty to be fair in their dealings with patients, colleagues and society and the dentist’s primary obligations include dealing with people justly and delivering dental care without prejudice. Accepting the principle of

justice in contemporary ethics is reflected in right to be treated. This right consists of three parts:

1. To be honest with patients
2. To give patients what they deserve
3. To give patients what they have right on.

**5. Veracity (truthfulness).** The dentist has a duty to communicate truthfully. This principle expresses the concept that professionals have a duty to be honest and trustworthy in their dealings with people and the dentist's primary obligations include respecting the position of trust inherent in the dentist-patient relationship, communicating truthfully and without deception, and maintaining intellectual integrity.

**6. Fidelity.** It is the obligation to keep implied or explicit promises.

**7. Confidentiality.** Dental professionals have a legal and ethical duty to keep patient information confidential. Principle of confidentiality means that a dentist must be discrete. Confidentiality comprises preservation of all information concerning patient, his/her diseases and treatment. Significance of confidentiality is in feeling of confidence a patient has, in belief that his/her doctor is "silent" despite everything heard or seen. It is the responsibility of dental professionals to treat any information about patients as confidential and only use it in the context in which it was given. Confidential information should be kept in a secure place at all times to prevent unauthorized or accidental disclosure.

Professional and ethical problems may be related to:

- diagnosis of diseases of the teeth;
- anesthesia;
- preparation of cavities;
- endodontic treatment;
- occupational health;
- local drug treatment.

**Diagnosis of diseases.** One of the most important functions of dentist correct diagnosis. However, the diagnosis may be correct inaccurate or erroneous. The professional duty of the physician to correct diagnosis of dental disease. At the heart of the ethical problems associated with incorrect diagnosis, the doctor is the professional level. The higher it is, the smaller the error, and vice versa.

**Anesthesia.** This feature is extremely important the dentist, as the painful surgery inhumane. This function is not always performed satisfactorily and therefore the majority of the population is afraid of dental procedures. In all civilized countries, all treatments in dentistry, which may be accompanied by tenderness, conducted under anesthesia.



*Dissection of the cavity.* The basis of possible ethical problems in dissection may be incorrect following steps: preparation painful, overheating of the tooth, injury to tissues of the mouth, accidental opening of the pulp cavity, the destruction of tooth structure, insufficient preparation.

*Filling.* Key ethical issues associated with this function dentist may arise in connection with the loss of seals. Often this happens in 2-3 days after the visit to the dentist. Correct to seal loss is due to substandard work dentist. The optimal solution is the recognition of their professional errors and improving the professional level.

*Endodontic treatment.* Professional and ethical issues arising from complications after endodontic procedures.

To prevent this kind of professional and ethical conflicts should:

- ✓ to master endodontic procedures;
- ✓ do not expose the patient at risk in cases of uncertainty about the success of treatment.

*Professional hygiene.*

- Roth dentist should be in perfect condition;
- You can not talk to the patient about the poor hygiene, without confirming it is an objective test;
- Discussion of oral hygiene should take place in a positive value.

*Local drug treatment.* Problems arise due to:

- Lack of information about new drugs dentist;
- Illiterate use of drugs;
- Complications in their application;
- Side effects of drugs.

Prevention of professional and ethical issues must be to maintain high professional competence of a doctor.

## **Question 2. Structure and equipment dental office.**

**The basic requirements are presented to him.**

*Sanitation requirements for the offices of therapeutic dentistry.* The area of the dental office should be 14 square meters per dental chair (height of 3.3 meters and a depth of 6 meters) for each additional - 7 square meters, in the event of a universal dental unit - 10 square meters.

*Finishing cabinet.* The walls in the dental office should be smooth, without cracks, ornaments, cornices, painted with oil paint or tiled to 2/3 height. Wall color should be pale shades. The ceiling of the cabinet is painted in white. Windows and doors are painted in bright colors. Paul's office should be covered with linoleum and have no gaps. Lighting in the dental office should be natural and artificial. Artificial lighting can be general and local.

The air temperature in the cabinet should be 22 degrees. Ventilation supply and exhaust.

*Equipping the dental office.* To equip the dental office is necessary equipment that division can be divided into groups:

1) The equipment and the equipment required to perform medical manipulations:

- ✓ Dental unit, dental chair, the chair of the doctor, the assistant chair, dental doctor's desk.
- ✓ Accessories: vitalometer, apex locator, diathermocoagulator and others.

2) Sterilizing equipment - drying ovens, glassperlen sterilizer, quartz lamp, and so on.

3) Equipment for nurses: a table for documentation, chair, computer and so on.

4) Facilities for processing and pre-treatment arms: a sink for hand washing and separately for tools, containers with disinfectant solutions.

5) Medical furniture: the cupboard of medicines, filling materials, couch, chairs for patients, kitchen sanitary equipment.

*Main blocks of the dental unit tools for manipulations in the mouth:*

- ✓ micromotors (speed 10,000-30,000 rpm)
- ✓ turbine handpiece (the rate of 300,000 - 500,000 rpm)
- ✓ other tools (skeler, curing light),
- ✓ spittoon,
- ✓ glass sink,
- ✓ saliva ejector,
- ✓ vacuum cleaner,
- ✓ water-air gun,
- ✓ dental chair,
- ✓ compressor,
- ✓ table dentist,
- ✓ chair dentist,
- ✓ chair assistant (15-20 cm above the chair of the doctor).

### **Question 3. Aseptic and antiseptic for dental seeing, definitely, main types.**

Asepsis is a system of preventive measures aimed at preventing the entry of microorganisms into the wound.

Asepsis involves performing disinfection, cleaning and sterilization of dental instruments, the use of personal protective equipment health workers special treatment doctor's hands.

Disinfection (decontamination) - a complex of measures aimed at the complete destruction of vegetative and dormant forms of certain kinds of microorganisms (spores and viruses remain) at the facilities of the environment in order to prevent transmission of the pathogen from infected to non-infected organism.

Disinfection physical methods.

- ✓ boiling in distilled water for 30 minutes after boiling;
- ✓ boiling in distilled water with the addition of 2% sodium bicarbonate for 15 minutes after boiling;
- ✓ effect of water vapor at a pressure in a steam sterilizer at  $t=110^{\circ}\text{C}$  for 20 minutes;
- ✓ effect dry hot air in an air sterilizer at  $t=120^{\circ}\text{C}$  for 45 minutes.

The chemical method of disinfection is the most common (e.g., immersion tool 6% hydrogen peroxide for 60 minutes).

*Presterilizing purification* consists of several stages. Upon completion of disinfecting instruments washed over a sink with running water for 30 seconds and are soaked in detergent solution (Inkrasept at a temperature of  $20-45^{\circ}\text{C}$ ) for 1 hour. Then, the washed medical instruments are rinsed under running water 3-10 min. Then medical instruments are rinsed under distilled water. The washed medical instruments are dried in a hot air oven at a temperature of  $85^{\circ}\text{C}$  until complete disappearance of moisture.

The quality of cleaning products from the blood is checked by setting the test of azopiram. The presence of residues of detergent products is determined by setting the phenolphthalein test.

*Antiseptic* - a set of chemical, mechanical, physical, biological methods to reduce the number of, suppression or total destruction of microorganisms in order to prevent the development of infection.

Chemical antiseptics carried out through the use of different antiseptics and antibiotics.

Biological antiseptic carried out by means of active or passive immunotherapy.

Mechanical antiseptic involves the use of mechanical methods to remove infected tissue or dental plaque.

Physical antiseptic is the use of physical methods of influence on the microorganisms with a view to their complete destruction.

#### **Question 4. Sterilization dental instruments, concepts.**

##### **Main types of sterilization used in clinic therapeutic dentistry.**

*Sterilization* is a complex of measures aimed at total destruction on the products or in products of all kinds of organisms, including their spore forms.

All medical products are used for manipulation in violation of the integrity of the skin and mucous membranes or in contact with the surface of the mucous membranes must be sterilized.

The sterilization of dental products is carried out physical and chemical methods.

*Sterilization of physical methods:*

- ✓ air - in air sterilizer at  $t=180^{\circ}\text{C}$  for 60 minutes, with  $t=160^{\circ}\text{C}$  for 150 minutes;
- ✓ steam - in a steam sterilizer at  $t=132^{\circ}\text{C}$  for 20 minutes, at  $t=120^{\circ}\text{C}$  for 45 minutes;
- ✓ in an environment heated balls - in glassperlen sterilizers intended for small dental instruments.

Sterilizers designed for small dental instruments using as sterilizing medium heated glass beads. Tools, sterilized in glassperlen sterilizer not be stored.

Shelf life of sterile instruments in a sealed package (to bikse, package kraft) is 3 days.

#### **Question 5. Key terms in the ergonomics dentists. Workplace of dentist.**

**Ergonomics** is the study of human performance and workplace design in order to maximize health, comfort and efficiency.

The design of the chair should allow the doctor to work sitting with the patient lying in the chair.

The materials of which made the chair should be required to withstand repeated handling antiseptics and disinfectants. There are desired colors of gentle tones.

The chair of the doctor and the assistant should be light and mobile, height adjustable products and seat back angle.

Suspended table doctor - «UNIT» (block) must be mobile and be located at "arm's length" and the order of arrangement of instruments determined by the order and the frequency of their use.

Depending on the arrangement of the modules «UNIT» installation are:

- *T-type*, «Dentsan», «Practic» (Chirana), «Ergostart 92 C» (Chirana), «Performer», «ADEC» (ADEC), «Cavo Systematica 1060 TK» (Cavo), «FimerFl Continental A» (Finland), «PM 2002 CC» (Planmeca OY), «BelDent (Belarus)», «Biotec CM 6-120» (Belarus), «Sirona Ml», «Spirit SI» (Siemens), when modules «Unit» arranged vertically;
- *S-type*, «Quint 7000», «FD-5000 Comfort» (Finndent), «Fimet Fl Continental E» (Finland), «ADEC international, Model 2080» (ADEC), «Cavo Systematica 1060 SK» (Cavo), «Promatic 1075 S» (Cavo), «Unident 1001» (JUGODENT), «Castellini», when modules are arranged horizontally;

- *G-type*, «Promatic 1075 G» (Cavo) modules mounted in the table, it is a fixed installation;
- *C-type*, «Cavo Junior» (Cavo), «Fimet Fl Cart» (Finland), «Unident 1002» (JUGODENT), «Ergostar 90 SU» (Chirana), the mobile unit with «Unit» operator.

### **Risk factors for dentist**

1. Repetition of performed task
2. Lack of rest/recovery between patients
3. Awkward postures during work
4. Contact stress
5. Vibrations from instruments
6. Poor workstation design and poor tools
7. Improper work habits
8. High forces needed to perform a task
9. Poor fitness level
10. Forceful exertions
11. Poor nutrition
12. Poor lighting

### **Characteristics of the main risk factors in the work of a dentist**

***Awkward Postures.*** More stress is placed on the spinal disks when lifting, lowering, or handling objects with the back bent or twisted compared with when the back is straight. Manipulative or other tasks requiring repeated or sustained bending or twisting of the wrists, knees, hips, or shoulders also imposed increased stresses on these joints. Activities requiring frequent or prolonged work over shoulder height can be particularly stressful. Dental personnel assume these awkward positions for the following reasons:

- To coordinate the relative positions between dentist and assistant.
- To obtain optimal view of teeth within the patient's mouth.
- To provide a comfortable position for the patient.
- To maneuver complex equipment and reach for instruments.

***Forceful Exertions.*** Tasks that require forceful exertions (like tooth extractions) place higher loads on the muscles, tendons, ligaments and joints. Prolonged experiences of this type can give rise to not only feelings of fatigue but may also lead to musculoskeletal problems when there is inadequate time for rest or recovery. Force requirements may increase with:

- Use of an awkward posture.
- The speeding up of movements.
- Use of small or narrow tool handles that lessen grip capacity.
- Increased slipperiness of the objects handled.
- Use of the index finger and thumb to forcefully grip an object (i.e. a pinch grip compared with gripping the object).

**Repetitive Motions.** If motions are repeated frequently and for prolonged periods, fatigue and muscle-tendon strain can accumulate. Effects of repetitive motions from performing the same work activities are increased when awkward postures and forceful exertions are involved. Repetitive actions as a risk factor can also depend on the body area and specific act being performed.

**Duration.** Job tasks that require use of the same muscles or motions for long durations increase the likelihood of both localized and general fatigue. In general, the longer the period of continuous work the longer the recovery or rest time required.

**Contact Stresses.** Repeated or continuous contact with hard or sharp objects, such as nonrounded desk edges or unpadded, narrow tool handles may create pressure over one area of the body (e.g., the forearm or sides of the fingers) that can inhibit nerve function and blood flow.

**Vibration.** Exposure to local vibration occurs when a specific part of the body comes in contact with a vibrating object, such as a power hand tool.

**Table 1. The main types of diseases that dentists suffer from**

Neck and Shoulder Disorders	<ul style="list-style-type: none"> <li>– Myofascial Pain Disorder</li> <li>– Cervical Spondylolysis</li> <li>– Thoracic Outlet Syndrome</li> <li>– Rotator Cuff Tendinitis/Tears</li> </ul>
Back Disorders	<ul style="list-style-type: none"> <li>– Herniated Spinal Disk</li> <li>– Lower Back Pain</li> <li>– Sciatica</li> </ul>
Hand and Wrist Disorders	<ul style="list-style-type: none"> <li>– DeQuervain's Disease</li> <li>– Trigger Finger</li> <li>– Carpal Tunnel Syndrome</li> <li>– Guyon's Syndrome</li> <li>– Cubital Tunnel Syndrome</li> <li>– Hand-Arm Vibration Syndrome</li> </ul>
Raynaud's Phenomenon	

#### The optimum operating position of the doctor and the patient

Supine positioning of the patient in the chair is usually the most effective way to help to maintain neutral posture. The chair should be raised so the operator's thighs can freely turn beneath the patient's chair. Clearance around the patient's head should at least allow unimpeded operator access from the 7 to 12:30 o'clock position, for right-handed operators. For most intraoral access sites, the maxillary plane should be extended 7° beyond the vertical. For treating the maxillary second and third molars, the maxillary

plane should be  $25^{\circ}$  beyond the vertical. For the mandibular anterior teeth, bring the patients chin down so the maxillary plane is  $8^{\circ}$  ahead of the vertical.

To sum up we can say that operating position of the doctor, with the patient lying on his back, will be the following:

- browsing,
- back straight.
- without bending.
- foot flat on the floor.
- body slightly tilted forward.
- upper arms are close to the body.
- a universal position for 12 hours.
- the distance from the eye doctor to the patient's mouth is 40 cm.

#### Position assistant dentist

Area of work is the assistant for 2-5 hours at 10 cm above the operator.  
Area transportation tools 5-8 hours.

#### The position of the patient

Most physiological, comfortable and is optimal posture of the patient when he is in the chair, and the tip of the nose and toes form a single line. Exceptions are patients with the following contraindications:

- pregnant women;
- aged people;
- patients who have problems with the spine;
- patients who have respiratory diseases;
- people absolutely do not want to be treated in this position.

Here are six keys to wellness to help a dentist to work more comfortably, with less fatigue and extend their career:

1. First and foremost, correct the ergonomic problems in the operatory.
2. Physical therapists, neuromuscular therapist should be consulted for musculoskeletal disorders.
3. Major trigger points should be resolved before any strengthening exercise is attempted.
4. Strengthen specific stabilizing muscles (like shoulder and back).
5. Be patient, but most of all commit to a regular regimen of prevention strategies.
6. Chairside stretching is an important strategy to perform throughout the workday to prevent microtrauma and muscle imbalances.

### **Tests to the topic**

**1. On the main chair in the dentist's office is needed area:**

- a. 20 square meters.
- b. 14 square meters.
- c. 10 square meters.
- d. 59 square meters.
- e. 13 square meters.

**2. If you have universal systems in the dental office need more space:**

- a. 14 square meters.
- b. 10 square meters.
- c. 7 square meters.
- d. 13 square meters.
- e. 59 square meters.

**3. Methods of disinfection are:**

- a. Physics, chemistry.
- b. The physical, the air.
- c. Chemical, steam.
- d. Steam, the physical.
- e. Steam, the air.

**4. The processing steps dental instruments are:**

- a. Disinfection, cleaning, sterilization.
- b. Disinfection, cleaning, boiling, sterilization.
- c. Autoclaving, pre cleaning, sterilization.
- d. Disinfection, autoclaving, boiling.
- e. Autoclaving, boiling, sterilization.

**5. Disinfection of dental instruments chemical method involves the use of:**

- a. 1% chloramine solution, 4% hydrogen peroxide solution.
- b. 3% chloramine solution, 6% hydrogen peroxide solution.
- c. 1% chloramine solution, 4% hydrogen peroxide solution.
- d. 4% hydrogen peroxide solution, 3% chloramine solution.
- e. 4% hydrogen peroxide solution, 6% hydrogen peroxide solution.

**6. Methods of sterilization of dental instruments are:**

- a. Steam, chemical, boiling.
- b. Steam, chemical, air.
- c. Air, steam, boiling.
- d. Steam, boiling.



- e. steam, boiling.

**7. Phenolphthalein sample is carried out to detect:**

- a. Residual blood.
- b. Residual chlorine compounds.
- c. Residues of detergent (alkaline components).
- d. Residual blood and residues of detergent.
- e. Residual blood and residual chlorine compounds.

**8. Choose a solution to clean the tips twice:**

- a. 4% solution of hydrogen peroxide.
- b. 70<sup>0</sup> alcohol.
- c. 3% chlorhexidine.
- d. 1% chloramine solution.
- e. 4% hydrogen peroxide solution.

**9. Ways of hiv transmission are:**

- a. Fecal-oral.
- b. Sex.
- c. Contact-household.
- d. Transplacental.
- e. Parenteral.

**10. Factors of transmission in the dental practice are:**

- a. Hand medical officer.
- b. Tools, instruments, equipment.
- c. Towels, door handles, faucets.
- d. Dosage medium.
- e. All answers are correct.

## LESSON 2. DIAGNOSTIC METHODS IN THERAPEUTIC DENTISTRY

The questions to be studied for the learning of the topic:

1. Basic methods of examination of dental patient
2. Schematic survey of dental patients.
3. Dental status at WHO.
4. Clinical diagnostic tests
5. Special research methods
6. X-ray examination
7. Laboratory diagnosis
8. The index score of dental diseases
9. Preparation of a dental treatment plan of the patient.

### **Question 1. Basic methods of examination of dental patient.**

All methods are divided into basic and advanced. The main methods are: clinical (survey, inspection) and instrumental (sounding, percussion). Additional methods include: physical (EDI X-ray diagnostics, laser diagnostics), laboratory (bacteriological method, biochemical methods, cytology, histology, virology).

### **Question 2. Schematic survey of dental patients.**

Complaints: on the therapeutic reception most common complaints:

- ✓ pain (nature, duration, irradiation, which provokes that takes),
- ✓ bleeding of the gums,
- ✓ halitosis,
- ✓ functions like disorders (speech, chewing),
- ✓ the presence of dental plaque,
- ✓ prevention inspection.

*History of life.* It is the patient's memories about the lifestyle, health and social factors that could affect it:

- Social and living conditions,
- Conditions of work (occupational hazard).
- Common diseases (cardiovascular, gastrointestinal, endocrine).

It is essential to HIV, infectious disease, tuberculosis, sexually transmitted diseases.

- whether the patient took drugs
- aggravating factors (alcohol, smoking)
- hereditary pathology,
- the frequency of visits to the dentist,
- features hygienic measures,

- -allergie.

*History of the disease* - the patient's memories about the causes, manifestations and course of the disease:

- How long it onset,
- What the patient relates the emergence of the disease,
- What held diagnostic and therapeutic measures and their effectiveness,
- About the exacerbation of the disease and their duration.

### **Question 3. Dental status at WHO.**

Dental status at the WHO include: extraoral (external inspection), intraoral (oral examination).

Intraoral examination examine: the lips, the eve of the mouth, mucous membrane of the cheeks. Hard and soft palate, tongue, floor of the mouth, the teeth.

## Examples of cards for the dental patient examination (WHO)



### Oral Health Questionnaire for Adults

Identification number	Sex		Location		
1. <div style="display: inline-block; width: 20px; height: 20px; border: 1px solid black; margin-right: 5px;"></div> <div style="display: inline-block; width: 20px; height: 20px; border: 1px solid black; margin-right: 5px;"></div> <div style="display: inline-block; width: 20px; height: 20px; border: 1px solid black; margin-right: 5px;"></div> <div style="display: inline-block; width: 20px; height: 20px; border: 1px solid black;"></div>	Male <input type="checkbox"/>	Female <input type="checkbox"/>	Urban <input type="checkbox"/>	Periurban <input type="checkbox"/>	Rural <input type="checkbox"/>
1                  4	1	2	1	2	3

2. How old are you today? \_\_\_\_\_  
(Years)

3. How many natural teeth do you have?

No natural teeth ..... ☐ 0

1–9 teeth ..... ☐ 1

10–19 teeth ..... ☐ 2

20 teeth or more ..... ☐ 3

4. During the past 12 months, did your teeth or mouth cause any pain or discomfort?

Yes ..... ☐ 1

No ..... ☐ 2

Don't know ..... ☐ 9

No answer ..... ☐ 0

5. Do you have any removable dentures?

	Yes	No
	1	2
A partial denture?.....	<input type="checkbox"/>	<input type="checkbox"/>
A full upper denture?.....	<input type="checkbox"/>	<input type="checkbox"/>
A full lower denture? .....	<input type="checkbox"/>	<input type="checkbox"/>

6. How would you describe the state of your teeth and gums? Is it “excellent”, “very good”, “good”, “average”, “poor”, or “very poor”?

	Teeth	Gums
Excellent .....	<input type="checkbox"/> 1	<input type="checkbox"/> 1
Very good.....	<input type="checkbox"/> 2	<input type="checkbox"/> 2
Good .....	<input type="checkbox"/> 3	<input type="checkbox"/> 3
Average .....	<input type="checkbox"/> 4	<input type="checkbox"/> 4
Poor.....	<input type="checkbox"/> 5	<input type="checkbox"/> 5
Very poor .....	<input type="checkbox"/> 6	<input type="checkbox"/> 6
Don't know .....	<input type="checkbox"/> 9	<input type="checkbox"/> 9

<b>7. How often do you clean your teeth?</b>		
Never .....	<input type="checkbox"/> 1	
Once a month .....	<input type="checkbox"/> 2	
2–3 times a month.....	<input type="checkbox"/> 3	
Once a week.....	<input type="checkbox"/> 4	
2–6 times a week.....	<input type="checkbox"/> 5	
Once a day.....	<input type="checkbox"/> 6	
Twice or more a day.....	<input type="checkbox"/> 7	
<b>8. Do you use any of the following to clean your teeth?</b> (Read each item)		
	Yes	No
	1	2
Toothbrush.....	<input type="checkbox"/>	<input type="checkbox"/>
Wooden toothpicks .....	<input type="checkbox"/>	<input type="checkbox"/>
Plastic toothpicks? .....	<input type="checkbox"/>	<input type="checkbox"/>
Thread (dental floss) .....	<input type="checkbox"/>	<input type="checkbox"/>
Charcoal .....	<input type="checkbox"/>	<input type="checkbox"/>
Chewstick/miswak.....	<input type="checkbox"/>	<input type="checkbox"/>
Other .....	<input type="checkbox"/>	<input type="checkbox"/>
Please specify .....	<input type="checkbox"/>	<input type="checkbox"/>
<b>9.</b>		
	Yes	No
a) Do you use toothpaste to clean your teeth .....	<input type="checkbox"/> 1	<input type="checkbox"/> 2
	Yes	No
b) Do you use a toothpaste that contains fluoride? ....	<input type="checkbox"/> 1	<input type="checkbox"/> 2
Don't know .....	<input type="checkbox"/> 9	
<b>10. How long is it since you last saw a dentist?</b>		
Less than 6 months .....	<input type="checkbox"/> 1	
6–12 months .....	<input type="checkbox"/> 2	
More than 1 year but less than 2 years.....	<input type="checkbox"/> 3	
2 years or more but less than 5 years .....	<input type="checkbox"/> 4	
5 years or more .....	<input type="checkbox"/> 5	
Never received dental care .....	<input type="checkbox"/> 6	
<b>11. What was the reason of your last visit to the dentist?</b>		
Consultation/advise.....	<input type="checkbox"/> 1	
Pain or trouble with teeth, gums or mouth.....	<input type="checkbox"/> 2	
Treatment/ follow-up treatment .....	<input type="checkbox"/> 3	
Routine check-up/treatment.....	<input type="checkbox"/> 4	
Don't know/don't remember .....	<input type="checkbox"/> 5	

<p><b>12. Because of the state of your teeth or mouth, how often have you experienced any of the following problems during the past 12 months?</b></p>						
	Very often	Fairly often	Some-times	No	Don't know	
	4	3	2	1	0	
(a) Difficulty in biting foods .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
(b) Difficulty chewing foods.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
(c) Difficulty with speech/trouble pronouncing words .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
(d) Dry mouth.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
(e) Felt embarrassed due to appearance of teeth.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
(f) Felt tense because of problems with teeth or mouth .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
(g) Have avoided smiling because of teeth.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
(h) Had sleep that is often interrupted .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
(i) Have taken days off work .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
(j) Difficulty doing usual activities..	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
(k) Felt less tolerant of spouse or people who are close to you.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
(l) Have reduced participation in social activities.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
<p><b>13. How often do you eat or drink any of the following foods, even in small quantities?</b> (Read each item)</p>						
	Several times a day	Every day	Several times a week	Once a week	Several times a month	Seldom /never
	6	5	4	3	2	1
Fresh fruit.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Biscuits, cakes, cream cakes .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Sweet pies, buns.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jam or honey .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Chewing gum containing sugar .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Sweets/candy.....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>



Lemonade, Coca Cola or other soft drinks.. <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>						
Tea with sugar ..... <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>						
Coffee with sugar ..... <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>						
(Insert country-specific items)						
<b>14. How often do you use any of the following types of tobacco?</b> (Read each item)						
	Every day 6	Several times a week 5	Once a week 4	Several times a month 3	Seldom 2	Never 1
Cigarettes .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Cigars .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
A pipe .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Chewing tobacco .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Use snuff .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Other .....	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Please specify _____						
<b>15. During the past 30 days, on the days you drank alcohol, how many drinks did you <i>usually drink per day</i>?</b>						
Less than 1 drink .....	<input type="checkbox"/> 0					
1 drink .....	<input type="checkbox"/> 1					
2 drinks .....	<input type="checkbox"/> 2					
3 drinks .....	<input type="checkbox"/> 3					
4 drinks .....	<input type="checkbox"/> 4					
5 or more drinks .....	<input type="checkbox"/> 5					
Did not drink alcohol during the past 30 days .....	<input type="checkbox"/> 9					
<b>16. What level of education have you completed?</b>						
No formal schooling .....	<input type="checkbox"/> 1					
Less than primary school .....	<input type="checkbox"/> 2					
Primary school completed .....	<input type="checkbox"/> 3					
Secondary school completed .....	<input type="checkbox"/> 4					
High school completed .....	<input type="checkbox"/> 5					
College/university completed .....	<input type="checkbox"/> 6					
Postgraduate degree .....	<input type="checkbox"/> 7					
(Insert country-specific categories)						
<i>That completes our questionnaire</i> <i>Thank you very much for your cooperation!</i>						
Year <input type="text"/> <input type="text"/>	Month <input type="text"/> <input type="text"/>	Day <input type="text"/> <input type="text"/>	Interviewer <input type="text"/> <input type="text"/>	District <input type="text"/> <input type="text"/>	Country <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/>	

# World Health Organization

## Oral Health Assessment Form

### for Adults, 2013

Leave blank	Year	Month	Day	Identification No.	Orig/Dupl	Examiner
(1) <input type="text"/>	(4) <input type="text"/>	(5) <input type="text"/>	(10) <input type="text"/>	(11) <input type="text"/>	(14) <input type="text"/>	(15) <input type="text"/>
<b>General information:</b>				<b>Sex</b> 1=M, 2=F	<b>Date of birth</b>	<b>Age in years</b>
(Name) _____				<input type="text"/> (18) (19) <input type="text"/>	<input type="text"/> (24) (25) <input type="text"/>	<input type="text"/> (26)
<b>Ethnic group</b> (27) <input type="text"/>	<b>Other group</b> (29) <input type="text"/>	<b>Years in school</b> (31) <input type="text"/>	<b>Occupation</b> (33) <input type="text"/>			
<b>Community</b> (geographical location) (34) <input type="text"/>	<b>Location</b> Urban (1) Periurban (2) Rural (3) <input type="text"/>					
<b>Other data</b> _____ (37) <input type="text"/>	<b>Other data</b> _____ (39) <input type="text"/>					
<b>Other data</b> _____ (41) <input type="text"/>	<b>Extra-oral examination</b> _____ (43) <input type="text"/>					

<b>Dentition status</b>  <table style="width: 100%; text-align: center;"> <tr> <td></td> <td>18</td><td>17</td><td>16</td><td>15</td><td>14</td><td>13</td><td>12</td><td>11</td><td>21</td><td>22</td><td>23</td><td>24</td><td>25</td><td>26</td><td>27</td><td>28</td> </tr> <tr> <td>Crown (45)</td> <td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td> </tr> <tr> <td>Root (61)</td> <td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td> </tr> <tr> <td>Crown (77)</td> <td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td> </tr> <tr> <td>Root (93)</td> <td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td> </tr> <tr> <td></td> <td>48</td><td>47</td><td>46</td><td>45</td><td>44</td><td>43</td><td>42</td><td>41</td><td>31</td><td>32</td><td>33</td><td>34</td><td>35</td><td>36</td><td>37</td><td>38</td> </tr> </table>		18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28	Crown (45)	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	Root (61)	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	Crown (77)	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	Root (93)	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>		48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38	<b>Permanent teeth</b>  <b>Status</b> 0 = Sound 1 = Caries 2 = Filled w/caries 3 = Filled, no caries 4 = Missing due to caries 5 = Missing for any other reason 6 = Fissure sealant 7 = Fixed dental prosthesis/crown abutment, veneer, implant 8 = Unerupted 9 = Not recorded
	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28																																																																																							
Crown (45)	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>																																																																																							
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<b>Periodontal status (CPI Modified)</b>  <table style="width: 100%; text-align: center;"> <tr> <td></td> <td>18</td><td>17</td><td>16</td><td>15</td><td>14</td><td>13</td><td>12</td><td>11</td><td>21</td><td>22</td><td>23</td><td>24</td><td>25</td><td>26</td><td>27</td><td>28</td> </tr> <tr> <td>Bleeding (109)</td> <td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td> </tr> <tr> <td>Pocket (125)</td> <td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td> </tr> <tr> <td>Bleeding (141)</td> <td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td> </tr> <tr> <td>Pocket (157)</td> <td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td> </tr> <tr> <td></td> <td>48</td><td>47</td><td>46</td><td>45</td><td>44</td><td>43</td><td>42</td><td>41</td><td>31</td><td>32</td><td>33</td><td>34</td><td>35</td><td>36</td><td>37</td><td>38</td> </tr> </table>		18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28	Bleeding (109)	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	Pocket (125)	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	Bleeding (141)	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	Pocket (157)	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>		48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38	<b>Gingival bleeding</b>  <b>Score</b> 0 = Absence of condition 1 = Presence of condition 9 = Tooth excluded X = Tooth not present  <b>Pocket</b>  <b>Score</b> 0 = Absence of condition 1 = Pocket 4–5 mm 2 = Pocket 6 mm or more 9 = Tooth excluded X = Tooth not present
	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28																																																																																							
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# World Health Organization

## Oral Health Assessment Form

### for Adults, 2013

<b>Loss of attachment</b>  <b>Severity</b> 0 = 0–3 mm 1 = 4–5 mm    Cemento-enamel junction (CEJ) within black band 2 = 6–8 mm    CEJ between upper limit of black band and 8.5 mm ring 3 = 9–11 mm   CEJ between 8.5 mm and 11.5 mm ring 4 = 12 mm or more   CEJ beyond 11.5 mm ring X = Excluded sextant 9 = Not recorded  * Not recorded under 15 years of age		<b>Index teeth</b>  17/16    11    26/27 (173) <input type="text"/> <input type="text"/> <input type="text"/> (175) (176) <input type="text"/> <input type="text"/> <input type="text"/> (178) 47/46    31    36/37	<b>Enamel fluorosis</b> <input type="text"/> (179)  <b>Severity</b> 0 = Normal 1 = Questionable 2 = Very mild 3 = Mild 4 = Moderate 5 = Severe 8 = Excluded (crown, restoration, "bracket") 9 = Not recorded (unerupted tooth)
<b>Dental erosion</b>  <b>Severity</b> <input type="text"/> (180)  0 = No sign of erosion 1 = Enamel lesion 2 = Dentine lesion 3 = Pulp involvement  <b>Number of teeth affected</b> (181) <input type="text"/> <input type="text"/> (182)	<b>Dental trauma</b>  <b>Status</b> <input type="text"/> (183)  0 = No sign of injury 1 = Treated injury 2 = Enamel fracture only 3 = Enamel and dentine fracture 4 = Pulp involvement 5 = Missing tooth due to trauma 6 = Other damage 9 = Excluded tooth  <b>Number of teeth affected</b> (184) <input type="text"/> <input type="text"/> (185)		
<b>Oral mucosal lesions</b>  <input type="text"/> (186) <input type="text"/> (189) <input type="text"/> (187) <input type="text"/> (190) <input type="text"/> (188) <input type="text"/> (191)  <b>Condition</b> 0 = No abnormal condition 1 = Malignant tumour (oral cancer) 2 = Leukoplakia 3 = Lichen planus 4 = Ulceration (aphthous, herpetic, traumatic) 5 = Acute necrotizing ulcerative gingivitis (ANUG) 6 = Candidiasis 7 = Abscess 8 = Other condition (specify if possible) 9 = Not recorded  <b>Location</b> 0 = Vermilion border 1 = Commissures 2 = Lips 3 = Sulci 4 = Buccal mucosa 5 = Floor of the mouth 6 = Tongue 7 = Hard and/or soft palate 8 = Alveolar ridges/gingiva 9 = Not recorded		<b>Denture(s)</b>  <b>Upper</b> <input type="text"/> (192) <b>Lower</b> <input type="text"/> (193)  <b>Status</b> 0 = No denture 1 = Partial denture 2 = Complete denture 9 = Not recorded	
<b>Intervention urgency</b> <input type="text"/> (194) 0 = No treatment needed 1 = Preventive or routine treatment needed 2 = Prompt treatment (including scaling) needed 3 = Immediate (urgent) treatment needed due to pain or infection of dental and/or oral origin 4 = Referred for comprehensive evaluation or medical/dental treatment (systemic condition)			

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### Question 4. Clinical diagnostic tests.

1. Probing
2. Palpation
3. Percussion
4. Determination of mobility.
5. Mechanical test (biting hard on the subject).

6. Follow the fistulous.
7. Drying.
8. Staining.
9. Temperature test.
10. Local anesthesia.

*Probing.* It is performed to determine the integrity of hard dental tissue using a probe. It allows to determine:

- presence of caries and its complications,
- presence and depth of the periodontal pocket.

*Percussion:* tapping a pen tool or tweezers for cutting edge or the chewing surface of the tooth (horizontal and vertical). Normally it is painless.

*Palpation.* Held by pressing index finger on the region of interest, or the capture of the entire stratum, or fold two fingers. You can define:

- pain
- any sharp bony prominences
- edema
- tumor
- tooth mobility
- consistency
- tissue turgor
- nodal status
- state of the musculoskeletal system
- separation of pathological periodontal pockets
- bleeding gums palpation

#### **Definition of mobility. Mechanical test.**

Held by biting hard on the subject, to diagnose diseases of the apical and marginal periodontium.

*Colouration.* A 2% solution of methylene blue is applied onto the dried portion of the tooth for 2-3 minutes. Then the mouth rinsed with water. Then determine the degree of staining. It is used for diagnosis of caries and non-carious lesions, determination of hygienic habits.

They used special cavities or caries detectors - markers to determine the pathological changed tooth tissues during dissection. They were offered by Professor Fusayama (Japan).

*Temperature test.* The test tool is heated with heating or gutta-percha and the flame is applied to the tooth in the middle third of the facial surface.

Test cooling jet Use cold water or a cotton chloroethyl on the shelf or the Ad Hoc refrigerants (eg, spray Coolan)

There are several types of reactions:

- there is no response (complete necrosis of the pulp, apical periodontitis, tooth cavity obliteration)

- just passing reaction (healthy tooth, carious lesions, dental caries)
- quickly passing reaction (hyperemia of the pulp)
- painful reaction from the cold, the long-term passes (acute serous pulpitis)
- pain from hot, not long passes (acute purulent pulpitis)
- long growing slowly passing pain (chronic pulpitis).

*Drying.* Most often used for the diagnosis of caries and non-carious lesions. When drying hard tissues of the tooth enamel healthy air jet has a shiny, smooth surface, and the expression in caries - matte and textured. Also, using this method can be determined, pour the fillings.

### **Question5. Special research methods.**

- electric pulp test
- transillumination
- X-ray examination
- laser immunofluorescence techniques
- definition of bioelectric potential

#### **Electric pulp test.**

Devices: PDE-1-PDE 2 (Russia), PULPOTESTER (Latvia), Digitest (USA).

The methodology of: a study carried out with the assistant. The doctor puts one passive electrode on the patient's forearm, active - on the tooth. For canines and incisors at the cutting edge for premolars - to the top of hill. It is also possible to the bottom cavity or filling. This is due to the fact that the largest number of data points in the dentinal tubules containing water and reduce the electrical resistance of hard tissues. Assistant to turn the knob - the potentiometer until the patient senses and captures this value. One should note-tit that unformed teeth have a higher value of EDI, so the differential diagnosis should be investigated eponymous tooth on the opposite side.

1. Intact tooth 2-6 mA
2. Caries 2-10
3. Flushing pulp 12-18
4. Acute pulpitis 20-30
5. Purulent pulpitis 30-60
6. Chronic pulpitis 40-60
7. Chronic gangrenous 60-90.
8. Chronic hyperplastic 50-70
9. Necrosis of the pulp (dry) 60-80
10. Pulp necrosis (wet) 100
11. Chronic apical periodontitis more than 100

*Transillumination.* For the diagnosis of approximal caries. In the darkroom fiber optic nakonech nickname blue light is placed behind the investigated tooth perpendicular to its axis. Healthy tissue you-look transparent, cavities - as brown shadow in the shape of a hemisphere.

*Laser diagnostics (fluorescence method).* The device DIAGNOdent (KaVo, Germany). The device comprises a laser diode (wavelength 650 nm) and a photodiode. Activating light is transmitted by an optical fiber to the tooth, length-nofokusny filter collects and transmits the excitement back to the long-wavelength fluorescence. The digital display shows the maximum intensity of luminescence at the time the EC-repetition. The device has a nozzle for the diagnosis of fissure caries and caries on smooth-surfaces.

### **Question 6. X-ray examination.**

Carried out for:

- Determination of cavities,
- With injuries of teeth, jaws,
- To determine the depth of the caries process,
- For differential diagnosis forms of caries and apical periodontitis,
- To identify the type of the pathological process in the marginal and apical periodontitis,
- To evaluate the quality endodontic treatment,
- To evaluate the efficacy of treatment of periodontal diseases marginal.

The main types of X-ray in restorative dentistry:

1. Pritselnye dental pictures.
2. Orthopantomography.
3. BITEWING-radiography (Bite pictures).

Sights dental images allows to obtain a detailed view of the interdental septum, furcations roots, periodontal ligament.

Orthopantomogram provides a cross-sectional image of the entire dentition used as a single functional complex, to plan and evaluate the treatment of marginal periodontal.

BITEWING-radiography (Bite shots) is used for the diagnosis of approximal caries.

The order of the X-ray:

1. The quality of radiographs (shadows, overlays, and so on)
2. Determine the type of snapshot (intraoral, extraoral and panoramic)
3. Determine the anatomy of teeth and group membership
4. Position, size, shape of the tooth crown, a contour violation, the presence of pathological ones it.

5. The contours of the tooth cavity and root canal, the severity of the coronal tooth cavity, length, and width in the root canal, the presence dentikley etc.
6. Periodontal gap - normal uniform illumination strip between cement and cortical plate, at a pathology it narrows or expands, or its integrity is violated.
7. Determination of bone structure. Evaluate the structure of the cortical plate, interdental partitions, state spongy substance. In the pathology can be observed atrophy, osteopenia, roses, destruction, osteonecrosis, sequestration.

Description pathological shadows:

1. Localization
2. The ratio of the various anatomical structures (bones, maxillary sinus, mandibular canal)
3. The number of (single or multiple)
4. The shape (round, oval, right, wrong, etc.)
5. The sizes (up to 5mm - granuloma, 5-8 mm - kistogranulema, 8 mm - a cyst)
6. The nature of contours (smooth or rough, clear or fuzzy, blurred and sharp, smooth and polycyclic).
7. Intensity (compared to the intensity of tooth tissues or cortical bone)
8. The nature of the structure of the shade (homogeneous or heterogeneous).

**Question 7. Laboratory diagnosis.**

It held primarily for diseases of the periodontal and GPRS.

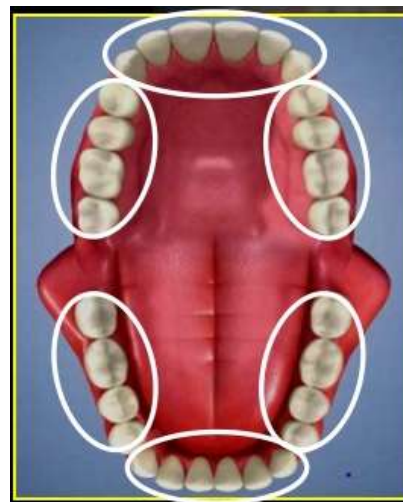
1. Bacteriological research: analysis of the flora obtained from the lesion.
2. Microscopic examination is carried out by light microscopy.
3. Molecular biological research methods (PCR and hybridization method).
4. Cytological methods (smear-imprint smear reprints, sediment washing liquid).
5. Histological examination (biopsy).
6. Virological studies (IFA, ELISA, infecting chicken embryos).
7. Allergic studies (intraoral tests in vivo, blood cell count, tests with a standard set of allergens).

**Question 8. The index score of dental diseases.**

1. For assessment of the oral hygiene (OHI-S, 1964).

**The Oral Hygiene Index-Simplified (OHI-S).** The six surfaces examined for the OHI-S are selected from four posterior and two anterior teeth.

- In the posterior teeth, the first fully erupted tooth distal to the second bicuspid, usually the first molar but sometimes the second or third molar, is examined on each side of each arch.
- In the anterior portion of the mouth upper right central incisor and lower left central incisor are scored.
- In the absence of either of these anterior teeth, the central incisor on the opposite side of the midline is substituted.
- Only fully erupted permanent teeth are scored. A tooth is considered to be fully erupted when the occlusal or incisal surface has reached the occlusal plane.
- Natural teeth with full crown restorations and surfaces reduced in heights by caries or trauma are not scored. Instead an alternate tooth is examined.



*Surfaces to be Seen.* Upper molars [6|6]: The buccal surfaces of selected teeth is inspected. Lower molars [6|6]: The lingual surfaces of the selected teeth are checked. Upper and Lower Central incisor 1: labial surface is scored.

Figure 1. Index Surfaces

Table 2. OHI-S Scoring Criteria

Scores	Debris	Calculus
0	No debris or stain present.	No calculus present.
1	Soft debris covering not more than one third of the tooth surface being examined or presence of extrinsic stains without debris regardless of surface area covered.	Supragingival calculus covering not more than one-third of the exposed tooth surface being examined.
2	Soft debris covering more than one third, but not more than two thirds, of the exposed tooth surface.	Supragingival calculus covering more than one-third but not more than two thirds of the exposed tooth surface and /or the presence of individual flecks of subgingival calculus around the cervical portion of the tooth.
3	Soft debris covering more than two thirds of the exposed tooth surface.	Supragingival calculus covering more than two-third of the exposed tooth surface or a continuous heavy band of subgingival calculus around the cervical portion of the tooth.

$$\text{OHI-S} = \frac{\text{Total debris score}}{\text{No. of segments scored}} + \frac{\text{Total calculus score}}{\text{No. of segments scored}}$$

**OHI-S is scored as follows:**

<i>WHO's interpretation</i>	<i>Interpretation in Republic of Belarus</i>
0.0–1.2 - Good oral hygiene	0,6 Good oral hygiene
1.3–3.0 - Satisfactory oral hygiene	0,7-1,6 Satisfactory oral hygiene
3.1–6.0 - Poor oral hygiene	1,7-2,5 Unsatisfactory oral hygiene
	> 2,6 Poor oral hygiene

2. For assessment of the gums condition (GI).

**Gingival Index (GI).** Attributed to Loe and Silness (1963), the GI assesses the severity of gingivitis based on color, consistency, and bleeding on probing. It describes the clinical severity of gingival inflammation as well as its location. The scoring is done on the entire dentition or on selected teeth. If it is done on selected teeth, then index teeth will be 16, 12, 24, 36, 32 & 44. Four gingival areas, i.e. distofacial, facial, mesiofacial and lingual surfaces are examined. A probe is used to press on the gingiva to determine its degree of firmness, and to run along the soft tissue wall adjacent to the entrance to the gingival sulcus.

*Teeth Examined:*

Maxillary right first molar

Maxillary right lateral incisor

Maxillary left first bicuspid

Mandibular left first molar

Mandibular left lateral incisor

Mandibular right first bicuspid.

*Surfaces Examined on each Tooth.* Distofacial, facial, mesiofacial and lingual surfaces.

*Scoring* is based on the following criteria.

**Table 3. GI Scoring Criteria**

<b>Scores</b>	<b>Criteria</b>
0	Normal gingiva/absence of inflammation
1	Mild inflammation: Slight change in color, slight edema. No bleeding on probing;
2	Moderate inflammation: Redness edema and glazing. Bleeding on probing;
3	Severe inflammation: Marked redness and edema. Ulceration and a tendency for spontaneous bleeding

Each surface is given a score, and then the scores are totaled which gives the score for area and divided by four gives score for the tooth. Totaling all scores and dividing by the number of teeth examined provides GI score per person.



*Interpretation:*

2.1–3.0. Poor (severe gingivitis), severe inflammation

1.1 – 2.0. Satisfactory (moderate gingivitis), moderate inflammation

0.1 – 1.0. Good (mild gingivitis), mild inflammation

< 0.1. Excellent (no gingivitis), no inflammation

The Belarusian index interpretation is similar to the WHO interpretation.

3. For assessment of the periodontal tissues condition (CPI, P.A. Leus, 1988).

**Complex periodontal index (P.A. Leus, 1988).** The complex periodontal index (CPI) represents the average value of a periodontal lesion signs: from risk factors (debris), early stages of the disease (bleeding, calculus) to developed stages (periodontal pocket, mobile teeth). The index is used for individual determination of periodontal status and mass surveys based on WHO's age groups. 17/16, 11, 26/27, 31, 36/37, 46/47 groups of teeth are examined in teenagers and adults. The definition of CPI during the examination of children under 3 years old and at the age of 5-6 years is not recommended.

**Table 4. CPI Scoring and Criteria**

Scores	Signs	Criteria
0	Healthy	Debris and signs of periodontal lesion during the examination are not determined
1	Debris	Any amount of soft white debris determined by a probe on the surface of the crown, in the interdental spaces or in the pre-gingival region
2	Bleeding	Bleeding during easy sensing of the periodontal groove (pocket)
3	Calculus	Any amount of hard deposits (calculus) in the subgingival area of the tooth
4	Pathological pocket	The periodontal pocket is determined by the probe.
5	Pathological mobility	Pathological tooth mobility of 2-3rd degree or absence of a tooth

$$\text{CPI}_{\text{individual}} = \frac{\text{Total score}}{\text{No. of segments scored}}$$

**Table 5. Levels of periodontal diseases intensity**

CPI	Levels of intensity
0,1-1,0	Risk of disease
1,1-2,0	Mild

2,1-3,5	Moderate
3,6-5,0	Severe

4. For assessment of the hard tissues of teeth condition (DFMT).

### **Decayed, Missing and Filled Teeth (DMFT) Index**

This index was developed by Henry Klein, Carrole E Palmer and Knutson JW in 1938. Based on the fact that the dental hard tissues are not self-healing and established caries leaves a scar. The tooth either remains decayed and if treated may be extracted or filled. It is an irreversible index.

DMFT describe the amount (the prevalence) of dental caries in an individual. DMFT numerically expresses the caries prevalence and is obtained by calculating the number of teeth (T) which are:

- Decayed (D)
- Missing (M)
- Filled (F).

It is thus used to get an estimation illustrating how much the dentition until the day of examination has become affected by dental caries.

Thus:

1. How many teeth have caries lesions (incipient caries not included)?
2. How many teeth have been extracted?
3. How many teeth have fillings or crowns?

*Selection of Teeth.* All 28 teeth are examined (based on 28 teeth).

Teeth not included are:

- ✓ Third molars
- ✓ Unerupted teeth (a tooth is considered as erupted when the occlusal surface or incisal edge is totally exposed)
- ✓ Supernumerary and congenitally missing teeth
- ✓ Teeth removed for reasons other than dental caries such as for orthodontic reasons and impactions
- ✓ Teeth restored for reasons other than dental caries, such as trauma, use as a bridge abutment and cosmetic purposes
- ✓ Retained primary tooth when the successor permanent is present.

The permanent tooth is considered.

*Procedure.* Each tooth is examined using a mouth mirror, an explorer and adequate light. The teeth should be observed by visual means as much as possible and only questionable small lesions should be checked by using an explorer.

### Rules for Scoring DMFT

1. No tooth should be counted more than once
2. Decayed (D), Missing (M) and Filled (F) teeth should be recorded separately
3. Tooth lost or filled due to reasons other than caries are not included

4. Deciduous teeth are not considered in DMFT index
5. A tooth with several filling is counted as one tooth.

#### Criteria for Recording

1. Decayed (D) recording:
  - When dental caries and a restoration are present on the same tooth, the tooth is recorded as D
  - When a crown is broken due to caries, it is recorded as D
  - Tooth with temporary restoration are recorded as decayed.
2. Missing (M) recording:
  - When a tooth has been extracted because of dental caries
  - When a tooth is carious, cannot be restored and is indicated for extraction.
3. Filled (F) recording:
  - Permanent restorations are recorded as F.

#### Criteria for Identification of Dental Caries

- Lesion is clinically visible and obvious
- Discoloration or loss of translucency typical of undermined or demineralized enamel
- Definite catch and the explorer tip can penetrate into soft yielding material.

#### DMFT Scores

The sum of the three figures forms the DMFT value. For example, DMFT of  $4 + 3 + 9 = 16$  means that 4 teeth are decayed, 3 teeth are missing and 9 teeth have fillings. It also means that 12 teeth are intact

#### Individual DMFT

Total each component separately ie total D, total M, total F.

$$\text{Total D} + \text{M} + \text{F} = \text{DMF SCORE}$$

#### Group Average

1. Total the D, M and F for each individual
2. Divide the total DMF by the number of individuals examined.

$$\text{Average DMF} = \frac{\text{Total DMF}}{\text{Total Number of Individuals Examined}}$$

#### Treatment Needs

$$\text{Percentage needing restorations (\%)} = \frac{\text{Total Number of D Tooth}}{\text{Total Number Examined}} \times 100$$

#### Limitations of DMFT Index

1. DMF values are not related to the number of teeth at risk. A DMF score does not directly gives an indication of the intensity of attack in any one

individual, e.g. a child of 8-year-old may have DMF score of 3 with only nine permanent teeth in mouth (one-third of teeth have been already affected by caries), whereas an adult may have a DMF score of 8 (more than the child score) out of 32 teeth (only one fourth of the teeth have been affected)

2. The DMF index is invalid when teeth have been removed or lost due to other reasons, e.g. periodontal reasons
3. The index gives equal weight to all the three components, i.e. missing decayed and well-restored teeth
4. Does not tell about the treatment needs of a person
5. The DMF index can overestimate caries experience in cases having teeth with preventive restorations
6. Cannot be used for root caries.

### **Question 9. Preparation of a dental treatment plan of the patient.**

Dental patient's treatment plan includes:

1. Emergency aid.
2. Motivation and training of oral hygiene.
3. Professional oral hygiene.
4. Selection of individual drugs and hygiene practices.
5. Therapeutic treatment.
6. Surgery treatment.
7. Orthopaedic treatment.
8. Medication treatment.
  - Local treatment.
  - General treatment.
9. Control visits 2 times a year

### **Tests to the topic**

#### **1. Which of the following diseases must be postponed sure to ask a dental patient?**

- a. Viral hepatitis.
- b. Colds.
- c. Tuberculosis.
- d. Venereal diseases.
- e. Hereditary diseases.

#### **2. Which of the following applies to intraoral examination?**

- a. Evaluation of the symmetry of the face
- b. Evaluation of the skin.
- c. Inspection of dentition.
- d. Inspection vestibule of mouth.

- e. Inspection of the oral mucosa.

**3. Most common complaints on the therapeutic reception are**

- a. Pain (nature, duration, irradiation, which provokes that takes).
- b. Bleeding of the gums.
- c. Halitosis.
- d. Functions like disorders (speech, chewing).
- e. The presence of dental plaque.
- f. Prevention inspection.
- g. All answers are correct.

**4. The methods of laboratory diagnostics include:**

- a. Microscopic examination.
- b. Virological examination.
- c. Probing.
- d. Biopsy.
- e. Percussion.

**5. Probing allows to determine:**

- a. Presence of caries and its complications.
- b. Presence and depth of the periodontal pocket.
- c. All answers are correct.

**6. Basic examination methods in therapeutic dentistry are:**

- a. Subjective.
- b. Objective.
- c. All answers are correct.

**7. Clinical diagnostic tests include:**

- a. Probing.
- b. Percussion.
- c. Staining.
- d. All answers are correct.

**8. The main types of radiographs used at a dental appointment are:**

- a. Targeted dental images.
- b. Orthopantomogram.
- c. BITEWING - X-ray.
- d. All of the above.

**9. Types of dental indexes are:**

- a. Oral hygiene indices.

- b. Periodontal indices.
- c. Dental indexes.
- d. All of the above.

**10. What can be determined during palpation?**

- a. Pain.
- b. Edema, tumor.
- c. Tooth mobility.
- d. State of the musculoskeletal system.
- e. Separation of pathological periodontal pockets.
- f. Bleeding gums palpation.
- g. All answers are correct.

### **LESSON 3. TREATMENT PLANNING IN THERAPEUTIC STOMATOLOGY**

The questions to be studied for the learning of the topic:

1. Preparation of a dental treatment plan of the patient.
2. Principles of treatment planning, depending on the clinical situation.
3. Basic methods of examination of dental patient.
4. Schematic survey of dental patients.
5. Dental status at WHO.
6. Clinical diagnostic tests.
7. Special research methods.

#### **Question 1. Preparation of a dental treatment plan of the patient.**

Dental patient's treatment plan includes:

1. Emergency aid.
2. Motivation and training of oral hygiene.
3. Professional oral hygiene.
4. Selection of individual drugs and hygiene practices.
5. Therapeutic treatment.
6. Surgery treatment.
7. Orthopedic treatment.
8. Medication treatment.
  - Local treatment.
  - General treatment.
9. Control visits 2 times a year.

#### **Question 2. Principles of treatment planning, depending on the clinical situation.**

1. The relief of pain
2. Removal of the necessary of teeth
3. The recommendations for the patient before treatment
4. Improvement of periodontal status
5. Treatment of carious teeth
6. Conducting endodontic treatment
7. Rational prosthetics.

**Table 6. Factors affecting treatment planning**

Related to the patient	<ul style="list-style-type: none"><li>– The confusion of anamnesis,</li><li>– Anxiety,</li><li>– The lack of time.</li></ul>
Related to the dentist	<ul style="list-style-type: none"><li>– The lack of qualification,</li></ul>

	– The lack of necessary equipments
Related to the cost of treatment	– The level of the patient's income – The availability of treatment for the patient
Others	– Not to talk about the treatment before full the survey, – To offer all variants, – Making changes during treatment, – Good oral hygiene, – Work in one segment of the oral cavity, – Use the simplest methods of treatment for anxious patients.

### **Question 3. Basic methods of examination of dental patient.**

All methods are divided into basic and advanced. The main methods are: clinical (survey, inspection) and instrumental (sounding, percussion). Additional methods include: physical (EDI X-ray diagnostics, laser diagnostics), laboratory (bacteriological method, biochemical methods, cytology, histology, virology).

### **Question 4. Schematic survey of dental patients.**

Complaints: on the therapeutic reception most common complaints:

- ✓ pain (nature, duration, irradiation, which provokes that takes),
- ✓ bleeding of the gums,
- ✓ halitosis,
- ✓ functions like disorders (speech, chewing),
- ✓ the presence of dental plaque,
- ✓ prevention inspection.

*History of life.* It is the patient's memories about the lifestyle, health and social factors that could affect it:

- Social and living conditions,
- Conditions of work (occupational hazard).
- Common diseases (cardiovascular, gastrointestinal, endocrine).

It is essential to HIV, infectious disease, tuberculosis, sexually transmitted diseases.

- whether the patient took drugs
- aggravating factors (alcohol, smoking)
- hereditary pathology,
- the frequency of visits to the dentist,
- features hygienic measures,
- -allergie.



*History of the disease* - the patient's memories about the causes, manifestations and course of the disease:

- How long it onset,
- What the patient relates the emergence of the disease,
- What held diagnostic and therapeutic measures and their effectiveness,
- About the exacerbation of the disease and their duration.

### Question 5. Dental status at WHO.

Table 7. Dental status at WHO

Extraoral inspection	Intraoral inspection
Posture and gait	The vestibule of the oral cavity: - small (5 mm) - average (5-10 mm) - deep (10 mm)
Face symmetry	Frenulums and buccal cords: strong (attached to the top of the nipple) average (at the base of the nipple) weak (in the transitional fold)
Skin Color, humidity, pathological eruptions, turgor, painfulness to palpation. Norm - pale pink, moderate humidity, easily taken into the fold and immediately cracked down, painless to palpation	Excretory ducts of the salivary glands - parotid (at 2 molar) - submandibular and sublingual (on the bottom of oral)
The red border of lips Norm: bright pink, moderately moist, cracks and pathological elements are missing, closed, cupid bow	The oral cavity Frenulum of the tongue Tongue (plaque asymmetry, tremor, teeth imprints) Tongue papillae (filiform, fungiform, foliaceous, grooved)
The corners of the mouth Norm: some are omitted, defeat elements are missing	Hard and soft palate (the color, submucosal layer is expressed, line A)
Lymphonodes Groups Size (0.5 - 1 cm.) The consistency (soft-elastic) The shape (rounded) The surface (smooth) Cohesion among themselves and with skin is missing Painfulness to palpation is missing	The bottom of oral. The venous network, the ducts of the salivary glands, color
Temporomandibular joint The degree of opening of the mouth Painfulness at the opening of the mouth Pathological noise, crunch, the clicking	The tonsils and back of the throat Hyperplasia of the tonsils 1 degree (1 \ 3) 2 degree (1/3 - 2/3)

	3 degree (halfway) The rear wall of the pharynx (the wet, smooth, with sporadic follicles)
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### ***Description of the elements of defeat of the oral mucosa***

1. Localization
2. The type of element (primary or secondary)
3. The size (diameter)
4. The surface (smooth, embossed)
5. Coloration
6. Boundaries
7. Attitude to the surrounding tissue (plus or minus tissue)
8. The presence of plaque (fibrinous, necrotic)
9. Consistency
10. The presence of background changes (hyperemia, cyanosis and so on)
11. Painfulness.

If there are several elements noted:

- quantity
- polymorphism
- propensity to merge
- Kebner's, Nikolsky's symptoms.

**Table 8. The quantity of saliva**

Hypoptyalism	Hypersalivation
<ul style="list-style-type: none"> <li>➤ feverish conditions</li> <li>➤ diabetes</li> <li>➤ neuro-psychiatric disorders</li> <li>➤ diseases of the salivary glands</li> <li>➤ receiving drugs</li> </ul>	<ul style="list-style-type: none"> <li>➤ smoking</li> <li>➤ receiving drugs</li> <li>➤ helminthic invasions</li> <li>➤ diseases of the oral mucosa</li> </ul>

### ***Description of the state of the gum***

1. Color (normal pale pink).
2. Contour (in front teeth are pointed, in premolars and molars are trapezoid). Changes of gingival contour (atrophic, crateriform, spherical, dissected, scalloped).
3. The surface (norm resembles orange crust, in the pathology is smooth, shiny).
4. Consistency (norm, it is elastic, and in pathology is friable).
5. Bleeding (norm not determined).
6. The position of gum (norm is on the enamel-cement border). At a pathology may be offset in the coronal direction (hyperplasia) or apical (recession).

**Table9. *Types of pathological pockets***

The depth of lesion	<ul style="list-style-type: none"> <li>➤ Gingival (no periodontal destruction)</li> <li>➤ Periodontal (violation of periodontal)</li> <li>➤ Supraperiosteal (pathologically altered tissues adjacent to the bone)</li> <li>➤ Intraosseous (pathologically altered tissues found inside bones)</li> </ul>
The quantity of surfaces of the teeth involved in the pathological process	<ul style="list-style-type: none"> <li>➤ easy (one surface)</li> <li>➤ combined (two or more)</li> <li>➤ complex (on all sides)</li> </ul>
The degree of involvement of furcation	<ul style="list-style-type: none"> <li>➤ 1 degree - violation of attachment in furcation</li> <li>➤ 2 degree - the disappearance of the attachment under the crown, but on one side</li> <li>➤ 3 degree - the probe completely passes</li> <li>➤ 4 degree - the tunnel is seen by the eye</li> </ul>

For evaluate of involvement of furcation uses a curved tube Nabers.

### ***Evaluation of condition of the teeth***

Bite – orthognathic, pathological.

### ***Stages of dental examination***

1. The quantity of missing teeth.
2. The quantity of filled teeth and assessment of the seals.
3. The presence of dentures in the oral cavity.
4. The integrity of the teeth (the presence of caries and its complications).
5. The size and shape
6. The change of the color (white, white-yellow, brown)
7. The shine of enamel (haze is at caries)
8. Mobility (three degrees).

### **Question 4. Clinical diagnostic tests.**

1. Probing
2. Palpation
3. Percussion
4. Determination of mobility.
5. Mechanical test (biting hard on the subject).
6. Follow the fistulous.
7. Drying.
8. Staining.
9. Temperature test.
10. Local anesthesia.

*Probing.* It is performed to determine the integrity of hard dental tissue using a probe. It allows you to determine:

- presence of caries and its complications,
- presence and depth of the periodontal pocket.

*Percussion:* tapping a pen tool or tweezers for cutting edge or the chewing surface of the tooth (horizontal and vertical). Normally it is painless.

*Palpation.* Held by pressing index finger on the region of interest, or the capture of the entire stratum, or fold two fingers. You can define:

- pain
- any sharp bony prominences
- edema
- tumor
- tooth mobility
- consistency
- tissue turgor
- nodal status
- state of the musculoskeletal system
- separation of pathological periodontal pockets
- bleeding gums palpation

#### **Definition of mobility. Mechanical test.**

Held by biting hard on the subject, to diagnose diseases of the apical and marginal periodontium.

*Colouration.* A 2% solution of methylene blue is applied onto the dried portion of the tooth for 2-3 minutes. Then the mouth rinsed with water. Then determine the degree of staining. It is used for diagnosis of caries and non-carious lesions, determination of hygienic habits.

They used special cavities or caries detectors - markers to determine the pathological changed tooth tissues during dissection. They were offered by Professor Fusayama (Japan).

*Temperature test.* The test tool is heated with heating or gutta-percha and the flame is applied to the tooth in the middle third of the facial surface.

Test cooling jet Use cold water or a cotton chloroethyl on the shelf or the Ad Hoc refrigerants (eg, spray Coolan)

There are several types of reactions:

- there is no response (complete necrosis of the pulp, apical periodontitis, tooth cavity obliteration)
- just passing reaction (healthy tooth, carious lesions, dental caries)
- quickly passing reaction (hyperemia of the pulp)
- painful reaction from the cold, the long-term passes (acute serous pulpitis)
- pain from hot, not long passes (acute purulent pulpitis)

- long growing slowly passing pain (chronic pulpitis).

*Drying.* Most often used for the diagnosis of caries and non-carious lesions. When drying hard tissues of the tooth enamel healthy air jet has a shiny, smooth surface, and the expression in caries - matte and textured. Also, using this method can be determined, pour the fillings.

### **Question5. Special research methods.**

- electric pulp test
- transillumination
- X-ray examination
- laser immunofluorescence techniques
- definition of bioelectric potential

#### **Electric pulp test.**

Devices: PDE-1-PDE 2 (Russia), PULPOTESTER (Latvia), Digitest (USA).

The methodology of: a study carried out with the assistant. The doctor puts one passive electrode on the patient's forearm, active - on the tooth. For canines and incisors at the cutting edge for premolars - to the top of hill. It is also possible to the bottom cavity or filling. This is due to the fact that the largest number of data points in the dentinal tubules containing water and reduce the electrical resistance of hard tissues. Assistant to turn the knob - the potentiometer until the patient senses and captures this value. One should note-tit that unformed teeth have a higher value of EDI, so the differential diagnosis should be investigated eponymous tooth on the opposite side.

1. Intact tooth 2-6 mA
2. Caries 2-10
3. Flushing pulp 12-18
4. Acute pulpitis 20-30
5. Purulent pulpitis 30-60
6. Chronic pulpitis 40-60
7. Chronic gangrenous 60-90.
8. Chronic hyperplastic 50-70
9. Necrosis of the pulp (dry) 60-80
10. Pulp necrosis (wet) 100
11. Chronic apical periodontitis more than 100

*Transillumination.* For the diagnosis of approximal caries. In the darkroom fiber optic nakonech nickname blue light is placed behind the investigated tooth perpendicular to its axis. Healthy tissue you-look transparent, cavities - as brown shadow in the shape of a hemisphere.

*Laser diagnostics (fluorescence method).* The device DIAGNOdent (KaVo, Germany). The device comprises a laser diode (wavelength 650 nm)

and a photodiode. Activating light is transmitted by an optical fiber to the tooth, length-nofokusny filter collects and transmits the excitement back to the long-wavelength fluorescence. The digital display shows the maximum intensity of luminescence at the time the EC-repetition. The device has a nozzle for the diagnosis of fissure caries and caries on smooth-surfaces.

### **Tests to the topic**

#### **1. What factors should be considered when planning treatment?**

- a. The confusion of anamnesis.
- b. The lack of necessary equipments.
- c. Making changes during treatment.
- d. The level of the patient's income.
- e. The availability of treatment for the patient.
- f. Good oral hygiene.
- g. All answers are correct.

#### **2. The patient's dental treatment plan includes:**

- a. Motivation and training of oral hygiene.
- b. Professional oral hygiene.
- c. Drug therapy.
- d. Surgical treatment.
- e. Orthodontic treatment.

#### **3. The main types of treatment include:**

- a. Etiotropic.
- b. Pathogenetic.
- c. Clinical.
- d. Symptomatic.

#### **4. Medication treatment consists of**

- a. Local treatment.
- b. General treatment.
- c. All answers are correct

#### **5. Principles of treatment planning depending on the clinical situation are**

- a. The relief of pain.
- b. Removal of the necessary of teeth.
- c. The recommendations for the patient before treatment.
- d. Improvement of periodontal status.
- e. Treatment of carious teeth.
- f. Conducting endodontic treatment.

- g. Rational prosthetics.
- h. All answers are correct.

**6. Indicate the factors influencing treatment planning related to the patient:**

- a. The confusion of anamnesis.
- b. Anxiety.
- c. The lack of time.
- d. All answers are correct.

**7. Indicate the factors influencing treatment planning related to the dentist:**

- a. The lack of qualification.
- b. The lack of necessary equipments.
- c. All answers are correct.

**8. Indicate the factors influencing treatment planning**

- a. Not to talk about the treatment before full the survey.
- b. To offer all variants.
- c. Making changes during treatment.
- d. Good oral hygiene.
- e. Work in one segment of the oral cavity.
- f. Use the simplest methods of treatment for anxious patients.
- g. All answers are correct.

**9. If there are several elements of the lesion of the oral mucosa, then note**

- a. Quantity.
- b. Polymorphism.
- c. Propensity to merge.
- d. Kebner's, nikolsky's symptoms.
- e. All answers are correct.

**10. How are pathological pockets distinguished?**

- a. The depth of lesion.
- b. The quantity of surfaces of the teeth involved in the pathological process.
- c. The degree of involvement of furcation.
- d. All answers are correct.

## **LESSON 4. DENTAL PATIENT CARD. RECORDING AND REPORTING DENTAL RECORDS. HOW TO FILL OUT.**

The questions to be studied for the learning of the topic:

1. Outpatients dental health card, a concept components. Other recording and reporting on the dental admission.
2. How to fill the dental patient card. Examination of the patient during the initial treatment. Transferred and concomitant disease.
3. Dental status, rules of filling
4. Preparation of a dental treatment plan of the patient.
5. Making diary visits.

### **Question 1. Outpatients dental health card, a concept components.**

#### **Other recording and reporting on the dental admission.**

Recording and reporting on the dental admission form number 037/u-10 "Leaf daily accounting work of a dentist (dental paramedic)"; form number 039/10-in "Diary of accounting work of a dentist (dental paramedic); form number 039-W/Y-10, "Summary of dental health of the patients with primary-increment; form number 043/10-in "Dental patient card" and others.

The form number 037/u-10 "Leaf daily accounting work of a dentist (dental paramedic)" has the following columns: date, time of reception of patients, the number of full years, the name, address, diagnosis (ICD code and description), treatment (view and description), type of visit, the key code of the group intact dentition indices.

The form number 039/10-in "Diary of accounting work of a dentist (tooth paramedic-foot) includes the following sections: the total number of visits, number of visits to primary, preventive work, the diagnosis of the completed treatment, therapeutic treatment.

The form number 043/10-in "Dental patient card" co-featured bout is the main document of primary medical documentation, legally binding and wearing auxiliary character, which is reflected and documented in the dynamics state of dental health of the patient, the process of diagnosis, treatment and prevention dental diseases.

Outpatient map consists of several sections:

- Data;
- "Taking into account the list of appointments and stress radiographic studies";
- "Examination of the patient in the primary treatment";
- "Prior consent to medical intervention";
- Journal of visits.

In the section "examination of the patient in the primary treatment" includes subsections:



- Complaints;
- State of general health with the words of the patient;
- External inspection;
- Stomatological status;
- Total treatment plan based on the results of the patient examination in primary increments.

**Question 2. How to fill the dental patient card. Examination of the patient during the initial treatment. Transferred and concomitant disease.**

Form 043/u - 10 conducted for each patient at each visit dentists.

Form 043/u - 10 is filled by a dentist in Russian or in legible handwriting on the computer. Filling in all the fields and subject lines, only in the case of the provision of emergency care to the patient is allowed to fill only those partitions which needed to reflect the process of emergency care. Filling the passport data held in the registry based on document of the patient.

Outpatient map is stored in the registry for 10 years after the last patient visit. Control over the conduct of the head of the patient card is carried out structural units.

Examination of the patient during the initial treatment. Transferred and comorbidities.

The primary visit is to visit a patient, first applied in the current year for dental care in this organization, regardless of the type of treatment. Any other visits to the dentist in the organization present year is repeated.

The string "Reason complaints" made the complaint of the patient and medical history data.

In the table, "the general state of health of the patient with the words" made data and related diseases, allergeanamneze, heredity, bad habits, occupational hazards, conditions of life.

In the column "delete as appropriate" designation "YES" crossed out on the diseases, whose presence at the patient denies; designation of "NO" crossed out on the diseases that the patient is at confirming or denying.

Under "If YES, specify" refined diagnoses identified diseases.

In the "other" information marked with a significant relation to withstanding the dental health of the patient.

In the "external examination" include data external examination of the patient: configuration persons, the state of the skin and red border, regional nodes, tic, temporomandibular joint.

**Question 3. Dental status, rules of filling.**

In the "dental status" include data on the state of the teeth, gums, periodontal, oral mucosa, oral hygiene.

Dental health noted in the appropriate boxes dentition conditional-governmental designations in the table.

- Status Designation tooth
- Healthy teeth 0
- Cavities 1
- Seal 3
- Removed 4
- Artificial crowns 7
- Impacted tooth 8
- Bridges 7 April 7

Status defined oral hygiene in tables «OHI-S» for children and ADULT-bite-integer constant.

Method for determining «OHI-S». Method for determining the CPI (A comprehensive periodontal index), GI (adult, child). To complex medical interventions in the provision of outpatient dental care include:

- invasive methods of diagnosis and treatment;
- ortopedic treatment;
- orthodontictreatment.

Juvenile persons found incompetent, the consent of their legal representatives provide, in the case of patients who are unable for health reasons to make an informed decision, the husband, and in its absence - a close relative. In the case of oral consent to medical intervention and refusal to issue it in writing in the line "give voluntary consent to medical intervention" word "intervention conducted by written consent of the patient or his legal representative, of" put the date, the signature of the attending physician and the head structural unit. Consent to medical intervention may be withdrawn, except when the doctor began to intervene.

#### **Question 4. Preparation of a dental treatment plan of the patient.**

Planning for dental treatment carried out on the basis of the first survey was conducted, and the diagnosis is individual, is an integrated, providing the unity of purpose of the medical activities. It presupposes a certain amount and the scheme of dental procedures. The treatment plan includes:

1. Emergency aid.
2. Motivation and training of oral hygiene.
3. Professional oral hygiene.
4. Selection of individual drugs and hygiene practices.
5. Non-surgical treatment.
6. Surgery.
7. Orthopedical treatment.
8. Orthodontic treatment.

9. Medication:
  - Local.
  - General.
10. Additional diagnostic measures.
11. Consultation other professionals.
12. Visits 2 times a year.

The physician should inform the patient or his legal representative with the treatment plan.

### **Question 5. Making diary visits.**

This section shall be entered information reflecting the dynamics of the process of providing assistance to dentists, indicating the date of visits, procedures carried out and the names of the physician with his signature. The attending dentist says complaints clinical picture, results of studies, makes a diagnosis. It makes a treatment plan and records of the treatment process. When you visit records are maintained until the end of treatment. On the department of therapeutic dentistry is available electronic version of the Order of the Ministry of Health of Belarus from 14.01.2011 № 24 "On approval of forms of primary medical documentation in dentistry."

### **Tests to the topic**

#### **1. The primary visit is considered:**

- a. Visiting a patient who first applied for dental care to this organization this year, regardless of the nature of the appeal.
- b. Any visit to the dentist in this organization in the current year.
- c. All answers are correct.

#### **2. The main sections of the dental outpatient card don't include:**

- a. Visit diary.
- b. Voluntary consent to medical intervention.
- c. Visual inspection.
- d. Passport data.

#### **3. The data of the external examination of the patient are indicated in the section:**

- a. Examination of the patient at the first visit.
- b. Voluntary consent to medical intervention.
- c. A diary of visits.

#### **4. Complex medical interventions in the provision of outpatient dental care include**

- a. Invasive methods of diagnosis and treatment.

- b. Ortopedic treatment.
- c. Orthodontictreatment.
- d. All answers are correct.

**5. What teeth are examined when determining the GI index:**

- a. 16, 24, 21, 41.44.36
- b. 11, 31, 16.36.46.26
- c. All teeth

**6. From what age can the CPITN index be determined:**

- a. From 3 years old.
- b. From 15 years old.
- c. From 19 years old.
- d. All answers are wrong.

**7. What score is given when determining the OHI-S index in the case of the presence of a pigmented dense plaque covering the entire tooth crown?**

- a. 0.
- b. 3.
- c. 1.
- d. 2.

**8. Are the teeth removed due to complicated caries taken into account in determining DMFT?**

- a. Yes.
- b. No.
- c. Not always.

**9. The visit diary notes:**

- a. Patient complaints.
- b. Diagnosis.
- c. Postponed and concomitant diseases.
- d. Dental formula.

**10. The treatment plan for a dental patient includes:**

- a. Emergency.
- b. Motivation and training in oral hygiene.
- c. Selection of individual means and methods of hygiene.
- d. Therapeutic treatment.
- e. All answers are correct.

## **LESSON 5. INDIVIDUAL AND PROFESSIONAL ORAL HYGIENE. THE INDICATION METHOD OF DENTAL PLAQUE.HYGIENIC INDEXES.**

The questions to be studied for the learning of the topic:

1. Dental deposits. Classification of dental deposits.
2. Non-mineralized dental plaque. The cuticle, pellicle, plaque. Definition, composition, mechanism of formation.
3. Types of calculus. Theories formation of calculus. Role in the pathology of the oral cavity.
4. Methods for detection of dental plaque and hygiene codes.
5. Individual oral hygiene.
6. Professional oral hygiene.

### **Question 1. Dental deposits. Classification of dental deposits.**

They are cuticle, pellicle by origin natural and microbial pellicle, dental plaque, tartar.

Localization all dental deposits are divided into: supragingival (pellicle, plaque, dental plaque, supragingival tartar) and subgingival (subgingival calculus).

According to the degree of infection all dental deposits are divided into: not infected (cuticle pellicle) and infected (dental plaque, dental plaque, tartar).

According to the degree of mineralization of all dental deposits are divided into: saline (tartar), non-mineralized (pellicle, plaque, dental plaque).

International Classification of Diseases (ICD-DA WHO, 1995)

- K03.6 Deposits [accretions] on teeth.
- K03.60 Pigmented dental deposits (black, green, orange).
- K03.61 Due to tobacco habit.
- K03.62 Due to betel-chewing habit.
- K03.63 Other gross soft deposits (Materia alba).
- K03.64 Supragingival calculus.
- K03.65 Subgingival calculus .
- K03.66 Dental plaque.
- K03.68 Other specified deposits on teeth.
- K03.69 Deposit on teeth, unspecified.

**Question 2. Non-mineralized dental plaque. The cuticle, pellicle, plaque.**  
**Definition, composition, mechanism of formation.**

*Cuticle* is the reduced epithelium of the enamel organ. Tooth loses cuticle shortly after the eruption of the tooth, so the clinical significance it has.

*Pellicle* is structureless uninfected acellular glycoprotein on the membrane surface in the tooth. It is a product of protein-carbohydrate complexes oral liquid, such as mucin glycoproteins, sialoprotein. The thickness is from 1 to 10 microns. The size of it is thinner on the crests perikemata thicker - in the furrows, on the contact surfaces of the tooth and gingival margin. Under the layer of plaque it thickens over carious spot becomes thinner.

*The role of the pellicle.* It controls the diffusion processes in the surface layer of enamel, teacher-exists acids on the tooth and the diffusion of calcium and phosphate from the tooth. It gives the enamel electingential permeability.

Distributed also felt that the pellicle gives rise to the formation of dental plaque. However Y. Erricson and B. Forsman reported that pellicle not only accelerates microbial colonization, and may even impede it. Possibly, pellicle plays a role in the development of caries (Leont'ev V.K., 1976), because regulates the diffusion and permeability in the surface layer of the enamel-prefecture, its solubility in an acidic environment. Changes in the composition and properties of the pellicle may favor the development of caries.

***Pellicle Formation.*** The initial attachment of bacteria begins with pellicle formation. The pellicle is a thin coating of salivary proteins that attaches to the tooth surface within minutes after cleaning. This layer is thin, smooth colorless and translucent and is called as acquired salivary pellicle. Initially pellicle is bacteria free. The function of salivary pellicle is mainly protective. Salivary glycoproteins and salivary calcium and phosphate ions are absorbed on to the enamel surface and this process may compensate for tooth loss due to abrasion and erosion. Pellicle also restricts the diffusion of acid products of sugar breakdown. It can bind other inorganic ions such as fluoride which promotes remineralization. The pellicle acts like double-sided adhesive tape, adhering to the tooth surface on one side and on the other side, providing a sticky surface facilitating bacterial attachment to the tooth surface. This layer is thin, smooth colorless and translucent and is called as acquired salivary pellicle. Following pellicle formation, bacteria begin to attach to the outer surface of the pellicle. Accumulation is greatest in sites which are protected from functional friction and tongue movement. The interdental region below the contact area is the site for greatest plaque accumulation. Bacteria connect to the pellicle and each other with hundreds of hair-like structures called fimbriae. Once they stick, the bacteria begin

producing substances that stimulate other free floating bacteria to join the community. Within the first two days in which no further cleaning is undertaken, the tooth's surface is colonized predominantly by gram-positive facultative cocci, which are primarily streptococci species. It appears that the act of attaching to a solid surface stimulates the bacteria to excrete an extracellular slime layer that helps to anchor them to the surface and provides protection for the attached bacteria. Within first few hours species of *Streptococcus* and a little later *Actinomyces* attach to the pellicle and these are the initial colonizers.

### Dental Plaque

Plaque can also be defined as the soft deposits that form the biofilm adhering to the tooth surface or other hard surfaces in the oral cavity, including removable and fixed restorations. Dental Plaque is a host-associated biofilm.

*Biofilms* are defined as "Matrix-enclosed bacterial populations adherent to each other and or/to surface or interfaces (by Costerton). According to the recent data (Widerer and Charaklis 1989), biofilm is defined as the relatively undefinable microbial community associated with a tooth surface or any other hard, non-shedding material.

*Dental plaque as a biofilm.* Structurally, dental plaque is now considered to be a biofilm of complex and dynamic microbial community. It contains areas of high and low bacterial biomass interlaced with aqueous channels of different sizes, which are the nutrient channels for bacterial colonization. The intercellular matrix forms a hydrated gel in which bacteria can survive and proliferate. Hence, biofilm adheres firmly to the tooth surface and is resistant to mechanical removal, as well as antibiotics.

Table 10. **Types of dental plaque**

<b><i>Supragingival plaque</i></b>	<b><i>Subgingival plaque</i></b>
<ol style="list-style-type: none"> <li>1. Coronal plaque, which is in contact with only the tooth surface/</li> <li>2. Marginal plaque, which is associated with the tooth surface at the gingival margin.</li> </ol>	<ol style="list-style-type: none"> <li>1. Attached plaque.</li> <li>2. Unattached subgingival plaque.</li> <li>3. Attached plaque can be tooth, epithelium and/or connective tissue associated.</li> </ol>

***Supragingival Plaque.*** It can be detected clinically only after it has reached a certain thickness. Small amounts of plaque can be visualized by using disclosing agents. The color varies from grey to yellowish-grey to yellow. The rate of formation and location of plaque vary among individuals and is influenced by diet, age, salivary factors, oral hygiene, tooth alignment, systemic diseases and host factors.

***Subgingival Plaque.*** It is usually thin, contained within the gingival sulci or periodontal pocket and thus cannot be detected by direct observation. Its presence can be identified only by running the end of a probe around gingival margin

***Tooth-associated Subgingival Plaque.*** The structure is similar to the supragingival plaque. The flora is dominated by Gram-positive cocci, rods, filamentous bacteria and some/few Gram-negative cocci and rods. This flora is associated with calculus formation, root caries and root resorption.

***Epithelium-associated Subgingival Plaque.*** This type of plaque is loosely adherent because it lacks the interbacterial matrix and is in direct association with the gingival epithelium, extending from the gingival margin to the junctional epithelium. This plaque predominantly contains Gram-negative rods and cocci, as well as a large number of flagellated bacteria and Spirochetes.

***Connective Tissue-associated Plaque.*** It is usually demonstrated in ANUG and localized aggressive periodontitis patients. The clinical significance is unclear. The unattached plaque can be seen anywhere. Thus, the tooth-associated subgingival plaque is most important in calculus formation, root caries and slowly progressive periodontal destruction, whereas unattached bacterial component is associated with rapid periodontal destruction.

### ***Composition of dental plaque.***

Bacteria + Intercellular matrix = Dental plaque

Bacteria make up approximately 70 to 80 percent of total material. One mg of dental plaque is estimated to contain 250 million bacteria. Other than bacteria, mycoplasma, fungi, protozoa and viruses may be present. The material among the bacteria in dental plaque is termed as intermicrobial/cellular matrix. It contains organic and inorganic portions. The organic matrix is composed of protein-polysaccharide complex produced by microorganisms. Carbohydrates in the form of levans (fructans) provides mainly energy while glucans (dextran) provide not only energy, but also act as the organic skeleton of plaque. Other carbohydrates are galactose and rhamnose. Glycoproteins provide the protein component and small amounts of lipids are also present. Inorganic components include, primarily calcium, phosphorus with small amounts of magnesium, potassium and sodium.

***Formation/development of dental plaque.*** Pellicle is the initial organic structure that forms on the surfaces of the teeth and artificial prosthesis. The first stage in pellicle formation involves adsorption of salivary proteins to apatite surfaces. This results from the electrostatic ionic interaction between hydroxyapatite surface which has negatively charged phosphate groups that interacts with opposite charged groups in the salivary macromolecules. The mean pellicle thickness varies from 100 nm at 2 hours to 500 to 1,000 nm.



The transition from pellicle to dental plaque is extremely rapid. The first components include mainly cocci with small number of epithelial cells and PMNL's, they form a monolayer within a few hours, and the attached bacteria proliferate and form small colonies of cocci. With time other types of microorganisms proliferate and form different microcolonies. Thus, in dental plaque development, two adhesion processes are required. First, bacteria must adhere to the pellicle surface and become sufficiently attached to withstand oral cleansing forces. Second, they must grow and adhere to each other to allow plaque accumulation.

***Bacterial Adherence.*** During initial adherence, interactions occur mainly between specific bacteria and the pellicle. They are:

*Bacterial Attachment via Electrostatic Interactions.* Oral bacteria bear an overall net negative charge, negatively charged components of the bacterial surface and negatively charged components of pellicle become linked by cations such as calcium.

*Bacterial Attachment via Hydrophobic Interactions.* These interactions are based on the close structural fit between molecules on the pellicle and bacterial surfaces. The nature of the hydrophobicity of the cell is not clearly known. The contributing factor might be lipoteichoic acid (LTA), which may provide a long hydrophobic area.

*Bacterial Attachment via Specific Lectin-like Substances.* Lectins in the bacterial surfaces recognize specific carbohydrate structure in the pellicle and become linked.

Adhesion and attachment occurs between:

- Bacteria and clean tooth surface
- Bacteria and pellicle
- Bacteria and same species
- Bacteria and different species
- Bacteria and matrix.

***Growth and Accumulation of Bacteria.*** Once the bacteria is adhered to the pellicle, subsequent growth leads to bacterial accumulation and increased plaque mass. Dental plaque growth depends on:

- a. Growth via adhesion of new bacteria
- b. Growth via multiplication of attached bacteria.

The initial bacteria that colonize the pellicle surface are mostly gram-positive facultative microorganisms such as *Actinomyces viscosus* and *Streptococcus sanguis*, as the plaque matures, secondary colonization of *Prevotella intermedia*, *Capnocytophaga*, *Porphyromonas gingivalis* takes place. This ability of bacteria to adhere to different species and genera of microorganisms is known as coaggregation.

### ***Structural and microscopic properties of plaque. Supragingival Plaque***

It is usually adherent to the tooth surface. It contains gram-positive cocci and gram-negative rods and filaments. The morphologic arrangement of the flora in supragingival plaque is described as “corn-cob” formations, characterized by a central core consisting of rod-shaped bacterial cells, e.g. *Fusobacterium nucleatum* and coccoid cells, e.g. streptococci which attach along the surface of the rod-shaped cell. The subgingival plaque differs from supragingival plaque, in that it contains many large filaments with flagella and is rich in Spirochetes. Tooth-associated plaque is similar to supragingival plaque; whereas tissue-associated plaque is covered with flagellated bacteria without a well-defined extracellular matrix and numerous bristle-brush formations. This arrangement is also called as “test tube brush” formation characterized by large filaments that form the long axis; and short filaments or gram-negative rods embedded in the amorphous matrix.

***Clinical significance of plaque.*** The microbial aggregations on the tooth surface if prevented from maturing may become compatible with gingival health. Supragingival plaque if allowed to grow and mature, may induce gingivitis and can lead to the formation of a microenvironment that permits the development of subgingival plaque. Therefore, supragingival plaque strongly influences the growth, accumulation and pathologic potential of subgingival plaque, especially in the early stages of gingivitis and periodontitis.

***Subgingival Plaque.*** In association with the presence of supragingival plaque, there are inflammatory changes that modify the anatomic relationships of the gingival margin and tooth surface. This results in enlarged gingiva, which increases the space for bacterial colonization and also protects bacteria from normal cleansing mechanisms. They derive nutrients from gingival crevicular fluid. Many of these microorganisms lack the adherence ability and utilize supragingival plaque bacteria as a means of colonization of the subgingival area. Electron microscopic studies have demonstrated the existence of an organic material called cuticle between the root surface and subgingival plaque. It is covered by a dense layer of microorganisms and is believed to be a remnant or secretory product of the junctional epithelial cells.

***Microbial specificity of periodontal diseases.*** Walter Loesche proposed the nonspecific and specific plaque hypothesis in 1976. The nonspecific plaque hypothesis states that it is the total bulk of plaque, which determines the pathogenicity rather than the individual species within it. In other words, all plaque is equally pathogenic. According to this, when only small amounts of plaque are present, the products released by this get neutralized by the host. Similarly, large amounts of plaque would produce large amounts of noxious products, which would overwhelm the host's defenses. However,

several authors have contradicted this concept. First, many patients have considerable amounts of plaque and calculus as well as gingivitis, but only a minority suffer from destructive periodontal disease even then in only few sites. This paradox might be explained by specific plaque hypothesis, which states that destructive periodontal disease is a result of specific microbial pathogens in plaque. Thus, although the amount of plaque present correlates well with disease severity, it correlates poorly in individual patients. But it is the nonspecific plaque hypothesis, which forms the basis for virtually all the current modalities for treatment, and prevention, which relies on the principle of reducing plaque scores to a minimum. Thus, although the nonspecific plaque hypothesis has been discarded in favor of the specific plaque hypothesis, much clinical treatment is still based on the nonspecific plaque hypothesis.

***Specific Plaque Hypothesis.*** It states that, not all plaque is pathogenic and its pathogenicity depends on the presence of certain specific microbial pathogens in plaque. This is based on the fact that, the specific microorganisms responsible for periodontal diseases release certain damaging factors that mediate the destruction of the host tissue. This concept was accepted easily due to the recognition of *Actinobacillus actinomycetemcomitans* as a possible pathogen responsible for localized juvenile periodontitis.

**Plaque pathogenic mechanisms.** The following are the possible pathogenic mechanisms by which the plaque microorganisms can cause periodontal disease.

- a. Physical nature of plaque.
- b. Invasion of tissues by bacteria.
- c. Release of toxic and inflammatory substances.
- d. Role of bacterial specificity.

### **Question 3. Types of calculus. Theories formation of calculus. Role in the pathology of the oral cavity.**

***Types.*** Depending upon the position of calculus in relation to the marginal gingival.

**Table 11. Calculus characteristic**

<b><i>Supragingival Calculus</i></b>	<b><i>Subgingival Calculus</i></b>
It is the tightly adherent calcified deposit that forms on the clinical crowns of the teeth above the free gingival margin. So, it is clinically visible. It is also called as salivary calculus because it forms from the saliva.	As the name implies, it is that calcified deposits that is formed on the root surfaces below the free marginal gingiva. It is believed to be formed from the gingival exudate and called serumal calculus

**Structure.** The deposits of supragingival calculus are usually whitish-yellow in color and can get stained by tobacco or food pigments, consistency is hard and clay-like. Since they derive the mineral salts from salivary secretions, they are most abundant on the lingual surfaces of lower anterior teeth, opposite Wharton's duct and Bartholin's duct and buccal aspects of maxillary molars opposite the Stenson's duct. Subgingival calculus is usually dark-brown or greenish-black in color and the deposits are firmly attached to the tooth surface. Since they are hard and firm, it cannot be removed easily. Unlike supragingival calculus, subgingival calculus can be found on any root surface with a periodontal pocket. Morphologically, it can appear in different forms, most commonly ring-like or ledge-like formations crusty, spiny or nodular deposits. Less frequently it can be seen as finger-like and fern-like formations.

**Composition.** It consists of inorganic and organic components Trace amounts of zinc, strontium, bromine, copper, manganese, gold and aluminium are also seen. At least, two-thirds of inorganic component is crystalline in structure. The main crystal forms are: Hydroxyapatite, Magnesium whitlockite, Octacalcium phosphate and Brushite.

**Attachment to the Tooth Surface.** Four types of attachment of calculus to tooth surface have been reported.

1. Attachment by means of an organic pellicle.
2. Mechanical interlocking into surface irregularities such as resorption lacunae and caries.
3. Penetration of calculus bacteria into cementum.
4. Close adaptation of calculus under surface depressions to the gently sloping mounds of the unaltered cementum surface.

Calculus when embedded deeply in cementum may appear similar in morphology and thus has been termed as calculocementum.

**Table 12. Differences between supra- and subgingival calculus**

<i>Indicators</i>	<i>Supragingival</i>	<i>Subgingival</i>
<b>Location</b>	Above the gingival margin	Deposits present below the margins of the gingiva
<b>Color</b>	White, yellow in color	Brown or greenish-black
<b>Source</b>	Derived from salivary secretions	Formed from gingival exudate
<b>Composition</b>	More brushite and octacalcium phosphate less magnesium whitlockite	Conversely less brushite and octacalcium phosphate and more magnesium whitlockite

<b>Salivary proteins</b>	Are present	Are absent
<b>Sodium content</b>	Is lesser	Increases with the depth of the pocket

**Formation of Calculus.** Calculus is nothing but, dental plaque that has undergone mineralization. Calculus is formed by the precipitation of mineral salts, which can start between 1st and 14th day of plaque formation. In two days plaque can be 50 percent mineralized and 60 to 90 percent gets mineralized in 12 days. Calcification starts in separate foci on the inner surface of the plaque. These foci of mineralization gradually increase in size and coalesce to form a solid mass of calculus. Calculus formation continues until it reaches maximum levels in about 10 weeks and 6 months, after which there is a decline in its formation, due to mechanical wear from food and from the lips, cheeks and tongue. This decline is referred to as reversal phenomenon.

**Table 13. Theories of Calculus Formation**

Precipitation of minerals can occur from a local rise in the degree of saturation of calcium and phosphate ions, this is explained in,	<p>a) <b><i>Booster mechanism</i></b>: according to this theory, precipitation of calcium phosphate salts results from a local rise in the pH of the saliva. Factors such as loss of carbon dioxide and production of ammonia could lead to rise in pH.</p> <p>b) <b><i>Colloidal proteins</i></b> in saliva bind to calcium and phosphate ions thus producing a supersaturated solution. When saliva stagnates in the oral cavity, colloids settle and result in the precipitation of calcium and phosphorous salts.</p> <p>c) <b><i>Phosphatase</i></b> liberated from dental plaque, desquamated epithelial cells, or bacteria precipitate calcium phosphate by hydrolyzing organic phosphates in saliva, thus increasing the concentration of free phosphate ions.</p>
“Epitactic Concept” (heterogenous nucleation)	According to this, seeding agents induce small foci of calcification. These foci enlarge and coalesce to form calculus. Hence, more appropriately called as heterogenous nucleation. The seeding

	agents in calculus is not clearly known, but suspected agents could be, intercellular matrix of plaque, carbohydrate protein complexes and plaque bacteria.
Inhibition theory:	This theory considers the possibility of calcification occurring only at specific sites because, there exists an inhibiting mechanism at non-calcifying sites. Wherever calcification occurs, the inhibitor is either altered or removed. One such inhibiting agents could be pyrophosphate which prevents the initial nucleus from growing, by possibly 'poisoning' the growth centers of the crystal.

Pathogenic Potential of Calculus in Periodontal Diseases. Before 1960s the belief was that calculus was the principle etiologic factor in periodontal diseases. However, the current view is that the initial damage to the gingival margin in the periodontal disease is due to the pathogenic effects of microorganisms in plaque. However, the effect could get more pronounced by calculus accumulation because it further provides retention of more plaque microorganisms. Therefore, there is no doubt that, the mineralized deposits can

- a. Bring the bacterial deposits more closely to the supporting structures.
- b. Interfere with the local self-cleansing defense mechanisms.
- c. And also enable the patients to perform proper oral hygiene methods.

#### **Question 4. Methods for detection of dental plaque and hygiene codes.**

1. Visual.
2. Staining.
3. Drying.
4. Probing.

One of the most informative methods for the detection of dental plaque is a painting. With this method you can determine the size, thickness, location of dental plaque.

To paint using special dyes, which can be divided into 2 groups: persistent (erythrosine) and unstable (iodine).

The method of application of the dye: dye is applied to the dried teeth with a cotton swab for 2 - 3 minutes, then allow the patient to rinse the mouth with water and evaluate the results. When the dental plaque is cisker, the intensity of the color greater.

On drying the tooth surface coated plaque has a roughened surface.

Probing for the detection of dental plaque was performed as follows: the probe is moved from the cutting edge or a knoll to the gum. In the case of dental plaque, it remains at the tip of the probe. With the sensing area can be determined plaque and the quantity.

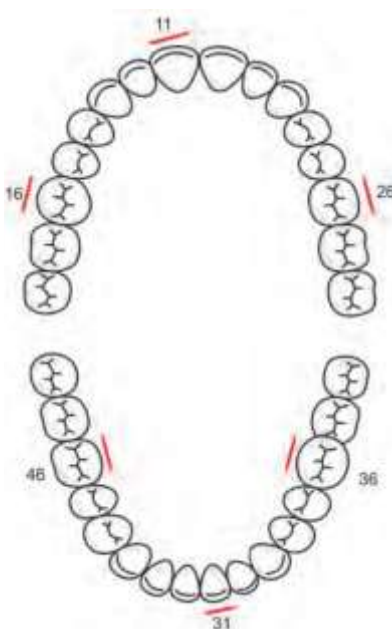
### **Determining the level of oral hygiene with sanitary codes.**

There are several groups of indices:

1. For assessment of the oral hygiene (OHI-S, 1964), Patient Hygiene Performance Index (PHP Index).
2. For assessment of the gums condition (GI; Loe, Silness, 1963).
3. For assessment of the periodontal tissues condition (CPI, P.A Leus, 1988; CPITN; WHO, 1982).

OHI-S, GI, CPI (P.A Leus) were discussed previously in topic 2. Consider in more detail the PHP index (Podshadley, Haley (1968) and the CPITN (WHO, 1982).

**Patient Hygiene Performance Index (PHP Index).** It was developed by Podshadley AG, and Haley JV (1968) to assess the extent of plaque and debris over a tooth surface as an indication of oral cleanliness. Debris for PHP was defined as the soft foreign material consisting of bacterial plaque, material alba and food debris that is loosely attached to tooth surfaces. Most useful for individual patients who have significant plaque accumulation.



*Figure 2. PHP index: 6 tooth surfaces are scored*

### **Tooth Numbers in FDI System**

16 - Upper right first molar

- 11 - Upper right central incisor
- 26 - Upper left molar
- 36 - Lower left first molar
- 31 - Lower left central incisor
- 46 - Lower right first molar

### **Surfaces**

Facial surfaces: incisors and maxillary molars.

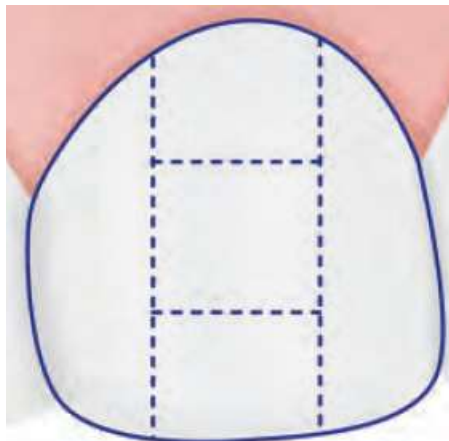
Lingual surfaces: mandibular molars.

### **Substitutions for Missing Teeth**

1. The second molar is used if the 1st molar
  - Is missing
  - Less than three-fourth erupted
  - Has a full crown
  - Is broken down/exposed surface area is reduced
2. The third molar is used when the second molar is missing.
3. The adjacent incisor of the opposite side is used, when the central incisor is missing.

### **Procedure**

- Disclosing solution is applied.
- Patient is asked to swish for 30 seconds and expectorate but not rinse.
- Examination is made using a mouth mirror.
- Each tooth surface to be evaluated is subdivided into five sections as follows



*Figure 3. Subdivision of a tooth into 5 sections (PHP index)*

*Vertically:* Three divisions mesial, middle and distal.

*Horizontally:* The middle third is subdivided into gingival, middle and occlusal or incisal thirds.

- Each area with plaque is scored a point so each tooth score can range from 1 to 5 points.



**Scoring.** Debris scores for individual tooth: Add the scores for each of the five subdivisions. The scores range from 0 to 5.

**PHP for an individual:** Total the scores for the individual teeth and divide by the number of the teeth examined. The PHP value ranges from 0 to 5.

**PHP Index for a group:** To obtain the average PHP score for a group or a population, total the individual score and divide by the number of people examined.

The index is calculated by the following formula:

$$\text{PHP} = \text{SUM CODES all teeth} / \text{number of examined teeth}$$

**PHPis scored as follows:**

<b><i>WHO's interpretation</i></b>	<b><i>Interpretation in Republic of Belarus</i></b>
Excellent - 0 (No debris)	0 - excellent level of hygiene
Good - 0.1–1.7	0.1-0.6 - good level of hygiene
Satisfactory - 1.8–3.4	0.7-1.6 - satisfactory level of hygiene
Poor - 3.5–5.0	1.7 and more - unsatisfactory level of hygiene.

**Community Periodontal Index of Treatment Needs (CPITN).** The CPITN is an epidemiologic tool developed by the World Health Organization (WHO) for the evaluation of periodontal disease in population surveys. It can be used to recommend the kind of treatment needed to prevent periodontal disease. Following extensive discussion and testing the CPITN was finalized and described in 1982. The CPITN is primarily a screening procedure which requires clinical assessment for the presence or absence of periodontal pockets, calculus and gingival bleeding. Use of a special CPITN periodontal probe (or its equivalent) is recommended. For epidemiologic purposes in adult populations, 10 specified index teeth are examined. For persons under 20 years of age only, six index teeth are specified. In dental practice, all teeth are examined and the highest score for each sextants noted. Only six scores are recorded. Measures of gingival recession, tooth mobility, intensity of inflammation, precise identification of pocket depths or differentiation between supra- and subgingival calculus are not included in the CPITN. Individuals are assigned to one of four treatment need categories determined from their CPITN scores.

Teeth examined: Two methods of selection

Sextants: Total six sextants

14 teeth on the maxilla and 14 teeth on the mandible, divided into three segments on each arch with following tooth numbers (FDI).

Maxilla:                      Mandible:

Sextant 1: 17 to      Sextant 4: 37 to

14	34
Sextant 2: 13 to	Sextant 5: 33 to
23	43
Sextant 3: 24 to	Sextant 6: 44 to
27	47

Third molars are not used unless they function in place of the second molars.

**Index Teeth.** In epidemiological surveys, for adults aged 20 years or more, only 10 index teeth are examined (5 teeth on the maxilla and 5 teeth on the mandible). These have been identified as the best estimators of the worst periodontal condition of the mouth.

MAX 17 16 11 26 27

MAND 47 46 31 36 37

The molars are examined in pairs and only one score, the highest is recorded. Only one score is recorded for each sextant.

For young people, up to 19 years only, six index teeth

MAX 16 11 26

MAND 46 31 36

The second molars are excluded as index teeth at these ages because of the high frequency of false (noninflammatory associated with tooth eruption) pocket.

For screening and monitoring purposes in dental practice all teeth in a sextant are examined for adults over age 19 years. Only one score, the highest is recorded for each sextant. When examining children less than 15 years, pockets are not recorded although probing for bleeding and calculus are carried out as routine.

*Recording Data.* The following box chart is recommended as the epidemiologic and dental office chart for recording CPITN data. The recommended periodontal probe for use with CPITN was described in the WHO 621 report (WHO 1978). The approved basic probe is suitable for general use in epidemiology and routine screening of patients in general practice. The CPITN is particularly designed for gentle manipulation of the often very sensitive soft tissues around the teeth; as such it is different in concept from the probes for dental caries and most other oral care instruments in current use.

*The Probe.* The probe is both thin in the handle and is of very light weight (5 gms). The probe has a black band starting at 3.5 mm and ending at 5.5 mm a ball tip of 0.5 mm diameter. The functions of ball tip are:

- To aid in detection of calculus and other tooth surface roughness.
- To facilitate assessment of the base of the pocket and reduce the risk of over measurement.

A variant of this basic probe has two additional lines at 8.5 mm and 11.5 mm from the working tip. The additional lines may be of use when performing a detailed assessment and recording of deep pockets for the purpose of preparing treatment plan for complex periodontal therapy. The two instruments can be identified as:

CPITN-E for the epidemiologic probe with a black band from 3.5 and 5.5 mm. CPITN-C for the clinical probe with the additional 8.5 and 11.5 mm markings

*Sensing Gingival Pockets.* An index tooth should be probed, using the probe as a “sensing” instrument to determine pocket depth and to detect subgingival calculus and bleeding response. The sensing force used should be not more than 20 grams. A practical test for establishing this force is to place the probe point under the thumb nail and press until blanching occurs. For sensing subgingival calculus, the lightest possible force that will allow movement of the probe ballpoint along the tooth surface should be used. The ball-end should be in contact with the root surface. When inserting the probe, the ballpoint should follow the anatomical configuration of the surface of the tooth root. If the patient feels pain during probing, this is an indicative of the use of too much force. The probe tip should be inserted gently into the gingival pocket and the depth of insertion read against the color coding. The total extent of the pocket should be explored and at least six points on each tooth should be examined: mesiobuccal, midbuccal, distobuccal, and the corresponding lingual sites.

*Codes and Criteria.* The codes are listed in the descending order of treatment complexity as follows:

Code X: When only one tooth or no tooth is present in the sextant (third molars are excluded unless they function in place of second molars).

Code 4: Pathological pocket of the 6 mm or more, that is, the black area of the CPITN probe is not visible. Note: If the designated tooth or teeth are found to have a 6 mm or deeper pocket in the sextant being examined, a code of 4 is given to the sextant. Recording of Code 4 makes further examination of that sextant unnecessary. There is no need to record the presence or absence of pathological pockets of 4 or 5 mm, calculus or bleeding.

Code 3: Pathological pocket of 4 or 5 mm that is when the gingival margin is on the black area of the probe. Note: If the deepest pocket is found at the designated tooth or teeth in a sextant is 4 or 5 mm, a code 3 is recorded. There is no need to examine for calculus or gingival bleeding.

Code 2: Calculus or other plaque retentive factors such as ill fitting crowns or poorly adapted edges of restoration are either seen or felt during probing. Note: The black band remains fully visible.

Code 1: Bleeding observed during or after probing (either immediate or delayed).

Code 0: Healthy tissue: The black band on the probe remains fully visible. There is no bleeding after probing. No calculus, restoration overhangs or other plaque retention factors are present.

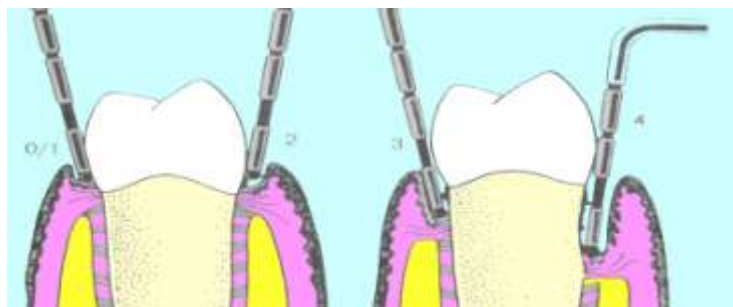


Figure 4. Community periodontal index of treatment needs

#### *Treatment Needs*

TN 0: A recording of code 0 (health) or X (missing) for all six sextant indicates that there is no need for treatment.

TN 1: A code of 1 or higher indicates that there is need for improving the personal oral hygiene of that individual.

TN 2: a. Code of 2 or higher indicates a need of professional cleaning of the teeth and removal of plaque retentive factors. Patient require oral hygiene instructions

b. Shallow to moderate pocketing (4 or 5 mm, code 3). Oral hygiene and scaling will reduce inflammation and bring 4 or 5 mm pockets to values of or below 3 mm. Thus, sextants of these pockets are placed in the same treatment category as scaling and root planning, i.e. Treatment Needs 2 (TN 2)

TN 3: A sextant scoring code 4 (6 mm or deeper pockets) may or may not be treated successfully by means of deep scaling and efficient personal oral hygiene measures. Code 4 is therefore assigned as complex treatment which can involve deep scaling, root planning and more complex procedures.

*Explanation of the Clinical Criteria and Treatment Needs.* Bleeding on gentle probing, plaque retentive factors (calculus or overhangs of restoration), 4 or 5 or 6 mm or deeper pockets are basic indicators if treatment needs. These criteria were chosen for the following reason:

1. TN 1: Bleeding is sign of an early disease which can be overcome by self-care following suitable oral health care educations and instructions. Control of gingival bleeding is a prerequisite for all periodontal therapy. This treatment is recognized as treatment need 1 (TN 1). The control or elimination of gingival bleeding should be the prime goal even if further treatment is not available.

2. TN 2: Although not pathological in themselves, Calculus and other plaque retentive factors favor plaque retention and inflammation. Unlike plaque that can be eliminated by self-care, the removal of calculus demands the professional care defined as treatment need 2 (TN 2).

3. TN 3: In patient with deep pocket even after scaling, root planning and control of bleeding by oral hygiene methods there will generally be residual pockets. The treatment of these conditions may require complex therapy for which skilled and trained dental professionals are needed. This treatment is recognized as TN 3.

### **Question 5. Individual oral hygiene.**

Individual oral hygiene is the elimination of plaque and food debris by a patient himself using personal hygiene products. It is impossible to eliminate completely the microorganisms of the oral cavity in the process of individual hygiene, it is possible only to reduce their number. Patient education and the development of oral care skills are challenging enough for both a doctor and a patient.

The problem includes patient motivation, education and instruction.

Education and instruction includes teaching a patient how to use various personal hygiene products using various techniques. One of the widely recommended methods for tooth brushing is the standard method:

1. The toothbrush is placed at an angle to the dentition at the point of gum attachment to the teeth. The plaque in the upper jaw is removed with sweeping movements from the gum downwards, and in the lower jaw is removed from the gum upwards. This is the way to brush external and internal surfaces of the teeth.

2. When cleaning the chewing surfaces of the teeth it is better to use horizontal movements.

3. When cleaning the incisors of the upper and lower jaws, the toothbrush is placed vertically.

4. It is recommended to clean the upper surface of the tongue in the direction from back to tip.

5. Rinse mouth with drinking water.

Every day the enamel on teeth is attacked by acids produced in dental plaque. These acids can make teeth weaker, and can result in decay. When fluoride reaches teeth, it is absorbed into the enamel. It helps to repair the enamel and prevent tooth decay. It can even help stop the decay process.

Fluoride can work from the outside of teeth, and from the inside of body. To work the best, you need to get it both ways. At home it is necessary to brush with fluoride toothpaste at least twice a day, especially after eating breakfast and before bedtime. If fluoride is greatest protection against decay, then frequent snacking can be teeth's biggest enemy. Every day, you face

snacking challenges. The truth is that what you eat isn't as important as when and how often you snack. It all has to do with the plaque reaction, and this is how it works. Everyone has plaque bacteria in their mouths. But when these plaque bacteria meet up with the sugars and starches that are found in snacks such as cookies, candies, dried fruits, soft drinks or even pretzels or potato chips, the plaque reacts to create acid, and a plaque attack occurs. The fact is most snacks that you eat contain either sugars or starches that give plaque this opportunity to make acid. And each plaque attack can last for up to 20 minutes after you have finished snack. During this period, the plaque acid is attacking tooth enamel, making it weak. That's when cavities can start! Sugars and sugary foods in the mouth are the main foods that germs (bacteria) thrive on to make acid which can contribute to tooth decay. Acid foods and drinks are also the main factor in tooth erosion. It is necessary to do following:

1. Limit the amount of sugary foods and drinks that you have. In particular, don't snack on sugary foods.

2. Try to reduce the amount of acid in contact with teeth. So, limit fizzy drinks (including fizzy water) and fruit juices as these tend to be acidic. Perhaps just limit yourself to one fizzy or fruit juice drink a day. Otherwise, choose drinks that are much less acidic, such as still water, and milk, tea, or coffee (without sugar).

3. Drink any acid drinks, such as fizzy drinks and fruit juices, quickly. Don't swish them around mouth or hold them in mouth for any period of time.

4. Brush teeth at least an hour after eating or drinking anything especially acidic foods and drinks.

5. Likewise, do not brush teeth within an hour of being sick (vomiting). This is because stomach acid will be part of the vomit.

By brushing twice daily with a fluoride toothpaste and by reducing the number of times you snack each day, you can help prevent tooth decay. When it comes to snacking, it's best to choose something nutritious and to snack in moderation. It's also better to eat the whole snack at one time! Here's why: eating five pieces of a snack at one time exposes teeth to possible tooth decay — for approximately 20 minutes. Nibbling on those same five pieces at five different times exposes teeth to possible tooth decay for approximately 100 minutes.

Limit between-meal snacking. Fewer snacks mean less acid exposure for teeth. If you snack, choose foods that are not fermentable carbohydrates.

**Best choices.** Cheese, chicken or other meats, or nuts. These foods actually may help protect tooth enamel. They do this by neutralizing acids or by providing the calcium and phosphorus needed to put minerals back in the teeth.

**Moderate choices.** Firm fruits such as apples and pears and vegetables. Firm fruits contain natural sugars. However, their high water content dilutes the effects of the sugars. These fruits also stimulate the flow of saliva, which fights bacteria and helps protect against decay. Vegetables do not contain enough carbohydrates to be dangerous.

**Worst choices** Candy, cookies, cakes, crackers, breads, muffins, potato, chips, french fries, pretzels, bananas, raisins and other dried fruits. These foods provide a source of sugar that certain bacteria can use to produce acid. The problem can be worse if the foods stick to teeth or get caught between them.

Four Steps to a bright smile:

1. Brush at least twice a day for about two minutes with a fluoride toothpaste, especially after eating breakfast and before bedtime.
2. Floss every day.
3. Limit the number of times you eat snacks each day.
4. Visit dentist regularly.

**TOOTHBRUSHING**

1. Place the toothbrush at a 45 angle along the gum line. Move the toothbrush toward occlusal surface by sweeping motion, and repeat for 10–12 times every 2 teeth.
2. Brush the inside surface of each tooth, using the same technique.
3. Brush the chewing surface (top) of each tooth.
4. Use tip of brush to brush behind each tooth - front and back, top and bottom and up and down strokes.

Firstly, brush the outer surfaces of the lower teeth. Then brush the inner surfaces of teeth in the same way as brushing the outer surfaces. When we brush the inner surfaces of front teeth, we should hold the toothbrush upright using gentle motion brushing from the gingival margin towards the crowns of the teeth. Then brush the chewing surfaces of the teeth with the toothbrush moving backward and forward. Finally, brush the outer surfaces, inner surfaces and the chewing surfaces of the upper teeth in the same way as the lower one.

Even if you have grasped the toothbrushing technique, it is important to use appropriate toothbrush and toothpaste. Toothbrushing cannot remove plaque from the proximal tooth surfaces. They have to be cleaned by dental floss, floss holder or interdental brush.

The order of brushing and flossing or interdental brushing does not affect the effectiveness of tooth cleaning. As long as you do these jobs thoroughly, you can achieve satisfactory results.

***Solutions to common problems encountered during toothbrushing.***

Areas that are hard to be reached by a toothbrush are difficult to be cleaned by toothbrushing, for example, the inner surfaces of lower teeth or crooked

teeth. The solution is to clean with a small head toothbrush or a single-tuft toothbrush. Some people may gag when they brush their teeth. To improve this situation, you can choose to use a toothbrush with a small head. The accumulation of dental plaque causes gingival inflammation. In that case, the gum will bleed when brushing. You can choose to use a toothbrush with soft bristles to clean away the plaque in such areas. Gingival inflammation will slowly subside and the gingivae will become healthy when the oral hygiene has improved.

**Manual toothbrush.** Shape of toothbrush head. All conventional toothbrush head designs are effective in cleaning every tooth surface. The tips of diamond shape toothbrush heads are narrower than those of the conventional ones. These tips are designed for easy access to posterior teeth. Brush head size should be approximately ~25 mm.

**Bristle pattern.** Toothbrush with block pattern has the bristles of the same length. They are arranged neatly like a block. Toothbrush with wavy or V-shape pattern is intended to give the bristles a better contact with the areas around the adjacent tooth surfaces. Multilevel trim pattern enables the brush to reach difficult-to-clean areas. Criss-cross pattern design can lift up plaque effectively. Medium or soft bristles are best for most people.

**Handle design.** All conventional toothbrushes have straight handles that are easier to control. Contra-angle handle design is similar to a dental instrument, intending to access to the difficult-to-clean areas. Flexible handle intends to reduce gum injury caused by excessive brushing force. Slip prevention grip handle intends to prevent the toothbrush from slipping away during toothbrushing. Some toothbrushes can be angled for increased access. Heat the neck under hot running water, bend to the desired angle and cool in cold water.

**Powered Toothbrush.** It has been shown that powered toothbrushes with a rotation oscillation action reduce plaque and gingivitis more than that of manual toothbrushes. However, to clean the teeth thoroughly, the most important is to adopt a proper and effective toothbrushing technique. For people with special needs, such as the physically and mentally disabled people, choosing powered toothbrushes may enhance the cleaning effect. Both manual and powered toothbrushes clean the teeth effectively as long as they are used correctly. Making sure you thoroughly clean teeth at least twice a day is more important than the type of brush you use.

The technique in using powered toothbrush is different from that of the manual toothbrush.

Electric toothbrush should have soft, nylon, and round-ended bristles for the most effective brushing. These bristles can wear with regular use and patient should inspect the brush regularly to maintain its integrity.



Hold toothbrush at a 45-degree angle to gum line. Keep the bristles in contact with tooth surface and gum line. It can help ensure to get the most effective brush possible. Only gentle pressure should be applied, as too much can injure teeth and gums. The vibrations of electronic toothbrush can also add a little additional pressure. Maintaining the 45-degree angle, brush the outer surfaces of 2–3 teeth using a back and forth rolling motion. The rolling motion is achieved by contacting the brush to the gum line and then moving downward with the toothbrush towards the chewing surface. To brush behind front teeth, tilt the brush vertically and make up and down strokes using only the front half of brush.

Use a gentle back and forth scrubbing motion to clean the biting surfaces and tongue.

Always rinse toothbrush with water after brushing. Store toothbrush in an upright position, if possible, and allow it to air-dry until using it again. Don't routinely cover toothbrushes or store them in closed containers, which can encourage the growth of bacteria.

Know when to replace toothbrush. Get a new toothbrush or a replacement head for electric or battery-operated toothbrush every three months, after contagious illness or sooner if the bristles are worn.

### **FLOSSING**

Toothbrushing cannot clean the adjacent surfaces of teeth. To remove the plaque accumulated on the adjacent tooth surfaces, we must use dental floss or interdental brush daily. Parents or caregivers can assist children or people in need to clean adjacent tooth surfaces by using a floss holder.

Plaque that is not removed can harden into tartar, which can only be removed through professional cleaning by a dentist. When this happens, brushing and cleaning between teeth become more difficult, and gum tissue can become swollen or may bleed. Regular flossing may reduce gum disease and bad breath by removing plaque that forms along the gum line.

Floss was once made from silk fibers twisted to form a long strand. Today, floss is usually made from nylon filaments or plastic monofilaments. Waxed, unwaxed, flavored, or plain floss all do the same thing. Waxed floss is coated with wax which makes it easier for the floss to slide into the interdental spaces. Mint flavoured floss gives a feeling of freshness. Fluoride coated floss is intended to prevent tooth decay from occurring on the adjacent tooth surfaces, but its effectiveness has not yet been proven.

**Shape of floss.** Flattened floss is designed to increase the contact surface with the tooth. Ultra floss is spongy and soft. Round floss is relatively thinner. Superfloss contains segments of stiffened-end threader, spongy floss and regular floss. Stiffened-end threader can make it easier for us to slide the superfloss through the gap between the teeth and fixed orthodontic appliances. Spongy floss cleans around the appliances and between wide

spaces or to floss underneath the bridge. Regular floss removes plaque from the adjacent tooth surfaces.

It's not what type of floss you use, but how and when you use it. If you have a preferred type of floss, you may be more likely to use it.

Other tools that may be used to clean between teeth include small brushes, special wooden or plastic picks, sticks or water flossers.

It is recommended to clean between teeth with floss (or another interdental cleaner) once a day. Some patients prefer to floss in the evening before bedtime so that the mouth is clean while sleeping. As long as you do a thorough job, it doesn't matter if you brush or floss first. However, flossing before brushing might allow more fluoride from toothpaste to reach between teeth. It is not recommended using a floss strand more than once. Used floss might fray, lose its effectiveness or may deposit bacteria in the mouth. Discard after use. Some people prefer floss tape which slides between teeth more easily than normal floss. Also, some people use disposable plastic forks with a small length of floss between the two prongs. These may be easier to hold and manipulate. However, they are expensive. The gums may bleed a little when you first begin to clean between teeth. This should settle in a few days. If it persists then it may indicate gum disease.

If you find it hard to handle floss, use an interdental cleaner — such as a special wooden or plastic pick, stick or brush designed to clean between the teeth.

### **Flossing technique**

1. Use a piece of dental floss which is approximately 45 cm long. Tie both ends to make a loop or wind most of the floss around the middle finger on one hand, and the rest around the middle finger on the other hand. Use the thumbs and the forefingers of both hands to hold the floss, leaving about 2 cm of floss in between.

2. Use a sawing or rubbing motion to slide the floss into the interdental space. Be gentle. Don't snap the floss into gums. When the floss reaches gumline, curve it into a C shape against one tooth.

3. Wrap around one tooth making a «C» shape and gently pull down to the deepest part of the gingival sulcus, and then slide it up and down with 8–10 strokes against the tooth. Then wrap around the adjacent tooth and repeat the up and down motions. Unwind fresh floss as you progress to the rest of teeth because used floss was contaminated with bacteria already. Clean all the other adjacent tooth surfaces in the same way.

**Floss holder (Dental floss stick).** Floss holder is a supplementary tool for flossing. It is suitable for parents or caregivers in helping children or individuals with special care needs to clean the adjacent surfaces of their teeth. There are many different types of floss holder in the market. One

should choose the appropriate type according to durability, shape, and handle length.

Floss holder comes in a «knife» shape or a «Y» shape. They are similarly effective in cleaning teeth. A new thread of dental floss can be reattached to the floss holder every time after use if it is a non-disposable one. When cleaning the back teeth using a «knife» shaped dental floss stick, one will need to stretch the lips to facilitate the access of the floss stick. Handles of floss holder differ in length. Those with shorter handles are more difficult to use. Therefore, parents and caregivers should choose the one with a longer handle to floss for their children or the elderly person.

To effectively clean adjacent surfaces of teeth, the floss on the floss holder must be taut. Therefore, we need to pay attention to the tautness of the floss when buying disposable floss holder.

**Method of using floss holder.** Move the floss holder left and right and slowly slide the floss towards the gingival margin. Pull the floss tightly against one of the adjacent tooth surfaces. Start from the deepest part of the gingival sulcus, gently slide the floss up and down to clean the adjacent tooth surface. Pull the floss tightly against the other adjacent tooth surface. Use the same technique, starting from the deepest part of the gingival sulcus, gently slide the floss up and down to clean the other adjacent tooth surface. Clean all the other adjacent tooth surfaces in the same way. If there is a wide gap between the neighboring teeth, an interdental brush may be used instead of dental floss.

### **INTERDENTAL BRUSHES**

Interdental brushes are the easy and effective way to clean between teeth. Interdental cleaning is the removal of plaque and impacted food from between the teeth, which normal toothbrush cannot reach. This is where a lot of dental disease starts. If the spaces between teeth are large like those teeth with gum recession due to gum disease, you may use interdental brush to clean the interdental space. Insert the interdental brush into the gap between the roots of the teeth, brush forward and backward to clean the adjacent tooth surfaces. Interdental brushes have small bristled heads specially designed to clean between teeth. The brush should fit snugly between the teeth. They are available from pharmacies and come in different width to suit the sizes of the gaps between teeth.

TePe Interdental Brushes are available in 9 colour-coded sizes. Extra soft brushes are available in 5 coded sizes. Another manufacturer produces four sizes of brushes. Generally, the spaces between the teeth at the front of the mouth are smaller than those at the back. Patient may need to use more than one brush size. The wires used in TePe Interdental Brushes are plastic coated and safe to use. It is recommended to change the brush every week or

when the bristles become worn or wires become buckled or distorted. Undue force and bending at severe angles will lead to damage of the wire.

Steps of use interdental brushes:

1. Use a brush appropriate for the size of the space between teeth.
2. Insert the brush gently between teeth — don't try to force the brush into the space. Insert the brush into the space between teeth at gum level, turning slightly. This technique aids access and prolongs the life of the brush. Once inserted, gently move the brush backwards and forwards a few times to remove plaque and debris.

3. If patient work to a pattern round the whole of mouth, it will help not to miss out any of the spaces.

Back of mouth: The new TePe Angle™ brush, with its long handle and preangled head, is ideal for cleaning difficult-to-reach areas such as between the teeth at the back of the mouth. The TePe Angle can be used to clean from both the tongue and cheek sides. Always rinse brush in clean water during and after use. When patient first start using interdental brushes, gums may be tender and bleed a little as you start to get rid of any plaque build-up. Carry on using the brushes and the bleeding should reduce as gums become healthier. If gums bleed on brushing, then it can be probably gum disease and patient needs periodontal treatment.

Interdental brushes TePe with the G2™ neck withstand well over 1000 repeated bending cycles. Thanks to the flexible neck, the brush is easier to use in the posterior area. Access is also extended, as the brush reaches further in between the teeth. This enables longer back-and-forth movements, which improve the cleaning effect. All sizes of TePe interdental brushes have plastic coated wire for safe cleaning. They clean implants and orthodontic appliances efficiently. The largest interdental brush can be used in difficult to reach areas: missing tooth or distal surface of the last tooth. For easier access curve the brush gently when cleaning the back teeth. Do not bend back. Use another straight brush for the front teeth. Interdental brushes TePe extra soft are recommended for delicate oral tissue or inflammation and for patients with mucosal sensitivity (sensitive gums and teeth, after oral surgery, dry mouth, mucositis, periimplantitis). They are also an excellent option for everyone who prefers a softer interdental brush.

TePe Proximal™ is recommended for those who prefer an interdental brush with a long handle. It facilitates cleaning between the posterior teeth and from the palatal and lingual sites. The color codes and sizes correspond with those of TePe Interdental brushes. The unique handcrafted dental model is a perfect visual tool for instructing patients in cleaning techniques using the best oral hygiene aid for every individual need. The color coded interdental spaces correspond the colours of TePe's interdental brushes. The dental model shows: crowding, furcation involvement, erupting molar and

missing tooth. If there is a wide gap between neighbouring teeth, an interdental brush may be used instead of dental floss. Insert the interdental brush into the gap between the teeth, placing the brush as close to the gum margin as possible, move the brush back and forth to clean the adjacent tooth surfaces.

It is rather difficult to clean the teeth with fixed orthodontic appliances in place. The teeth and the appliance should be cleaned in the morning and before bed at night every day, as well as after each meal. Replace with a new brush when the bristles are worn or deformed.

**SINGLE-TUFT TOOTHBRUSH.** It is a toothbrush with a very small head which can be used to clean wisdom teeth and crooked teeth effectively. There are various shapes of brush head. The effectiveness of cleaning by different shapes of the brush head is similar.

**PERIO-AID.** Perio-Aid is a toothpick in the holder. It is suitable for cleaning root surfaces near gingival cleft, where single-tufted brush cannot reach.

**CLEANING A FIXED BRIDGE.** As dental plaque also accumulates on the surface under the bridge, people wearing bridges should use superfloss to clean the area daily. Firstly, insert the stiffened end into the area between bridge and the tooth, then pull the superfloss until the spongy part reaches the bridge. Wrap the spongy floss around the real tooth, slide up and down to clean the adjacent tooth surface. Gently move the spongy floss between the gum and the base of the bridge, use a soft back-and-forth motion to remove plaque under the false tooth. Then wrap the spongy floss around the real tooth at the other side of the bridge, slide up and down to clean the adjacent tooth surface. Finally, slowly pull it out from the space between the tooth and the bridge. The regular floss is used to remove the plaque from the adjacent tooth surfaces for the rest of the teeth.

**CLEANING IMPLANT SUPPORTED SINGLE CROWN.** The cleaning method is similar to that of a real tooth. Brush at the gum margin, and use floss or interdental brush to clean the interdental surfaces.

**CLEANING IMPLANT SUPPORTED BRIDGE.** If the false teeth have two or more implants supporting, the cleaning method is similar to that of a conventional bridge. Since there is a gap between the false teeth and the gum, it is necessary to use superfloss or interdental brush to clean the surfaces underneath the false teeth.

**CLEANING IMPLANT SUPPORTED DENTURE.** Implant supported false tooth can help to improve chewing ability. However, if oral self-care is not appropriate, the gum around the implant may become inflamed, leading to development of peri-implantitis. Gradually, the implant may become mobile and even come out. After removing the denture, use a single-tuft toothbrush to clean around the abutment of the implant. The

cleaning method for the denture is the same as for conventional dentures. Use soft toothbrush and detergent to clean every part of the denture. Then rinse with water and immerse it in a glass of water overnight.

**CLEANING FIXED ORTHODONTIC APPLIANCES.** People wearing orthodontic appliances should clean the appliances every day as dental plaque also adheres to the appliances. It is rather difficult to clean the teeth with the fixed orthodontic appliance in place. People wearing fixed orthodontic appliances should pay special attention to their oral hygiene to reduce the chance of developing tooth decay or gum disease. They should clean teeth and the appliance every day in the morning and before bed at night as well as after each meal.

1. Firstly, brush the area between the orthodontic appliance and gingiva (gums). Then, brush the area between the appliance and the crown of the teeth.

2. Finally, insert an interdental brush into the area between the tooth and the appliance to clean.

Flossing is difficult for people wearing fixed orthodontic appliance. Hence, superfloss can be used to facilitate the removal of dental plaque in the adjacent tooth surfaces.

- 3 Push the stiffened end through the space between the teeth and the orthodontic appliance.

- 4 Then wrap the floss ends around middle fingers, hold it tightly between the thumbs and forefingers of both hands, leaving about 2 cm of floss in between. Gently pull the floss into the interdental space by using a sawing motion.

- 5 Wrap the floss around one tooth in a «C» shape, pull down to the deepest part of the gingival sulcus, and then slide up and down to clean the tooth surface. Then wrap around the adjacent tooth and repeat the cleaning.

**TOOTHPASTE.** The main function of toothpaste is to help remove dental plaque. Their various functions depend on the active ingredients they contain. The followings are the common types of toothpaste: fluoride toothpaste, desensitizing toothpaste, anti-calculus toothpaste, anti-plaque toothpaste, and whitening toothpaste. A pea-sized blob of toothpaste is adequate.

***Anti-decay toothpaste.*** They contain Fluoride Compounds such as Sodium Fluoride (NaF), Stannous Fluoride (SnF<sub>2</sub>), or Monofluorophosphate (MFP<sub>2</sub>) etc. It's important to use a toothpaste with the right concentration of fluoride. Check the packaging to find out how much fluoride each brand contains. Adults should use a toothpaste that contains at least 1000–1500 parts per million (ppm) fluoride. After brushing, spit out any excess toothpaste. Don't rinse mouth immediately after brushing, as it will wash

away the concentrated fluoride in the remaining toothpaste, thus diluting it and reducing its preventative effects.

Children toothpaste contains 500 ppm fluoride and is usually flavoured, e. g. fruit flavour, candy flavour, which is more appealing to children.

Since we are all susceptible to tooth decay, we all need fluoride toothpaste. Fluoride strengthens teeth, and increases their resistance to acid attacks. It inhibits the growth of dental plaque and reduces its acid-producing capability.

During the initial stage of tooth decay, when minerals are lost from the surfaces of the tooth, and a cavity has not formed yet, an appropriate amount of fluoride can promote the remineralization (enhance the saliva to replenish the lost minerals) of the tooth and repair early tooth decay.

***Desensitizing toothpaste.*** The active ingredients provide relief from dentine hypersensitivity symptoms in 2 ways. First, they interrupt the neurone response to pain stimuli; second, they occlude the dentinal tubules of dentine. Active ingredients such as Potassium Nitrate or Arginine, etc., are used by different product manufacturers. There are many different types of desensitizing toothpastes marketed by different brand names. Their various functions depend on different active ingredients they contain.

***Anti-calculus toothpaste.*** The active ingredient is Pyrophosphate or Zinc Citrate, etc. However, these inhibitors are not capable of dissolving existing deposits. Moreover, the effects of these anti-tartar products are mostly limited to the supragingival area.

***Anti-plaque toothpaste.*** This kind of toothpaste inhibits plaque accumulation, reduces effects of the bacterial toxins on the tooth surrounding tissues, thereby reduces the chances of getting gum disease. In the market, different anti-plaque toothpastes contain different active ingredients. For example, Triclosan or Zinc Citrate, Chlorhexidine Gluconate etc. It should be used no longer than 3 weeks.

***Whitening toothpaste.*** This kind of toothpaste contains relatively coarse abrasives which function by abrading the stains on the tooth surface, giving a whitening effect or enzymes. The effects of the long term use of this kind of toothpaste are still unknown.

## **MOUTHWASHES**

Depending on the active ingredients they contain, different types of mouthwash can be used to prevent tooth decay, reduce formation of dental plaque and inflammation of the gums or reduce tooth sensitivity.

***Fluoride mouthwash.*** This kind of mouthwash contains fluoride compounds such as 0.05 % Sodium Fluoride (NaF) which provides extra fluoride to the people who need it. Using it daily may give additional protection against tooth decay.

Don't use mouthwash — even a fluoride one — straight after brushing teeth or it will wash away the concentrated fluoride in the toothpaste left on teeth. Choose a different time to use mouthwash, such as after lunch. Don't eat or drink for 30 minutes after using a fluoride mouthwash.

For those who are prone to tooth decay or having severe tooth decay, wearing orthodontic appliance or after undergoing radiotherapy to the head and neck region, fluoride containing mouthwash can offer additional protection against tooth decay.

**Anti-plaque mouthwash.** It inhibits plaque accumulation, thus reducing the chance of getting gingivitis. The active ingredients include Chlorhexidine Gluconate, Triclosan, Thymol, Cetylpyridinium Chloride (CPC), etc. However, long term use of mouthwash may stain the teeth and alter taste sensation.

**Desensitizing mouthwash.** This kind of mouthwash contains active ingredients such as Arginine which claims to seal the dentinal tubules at the sensitive site, thus reducing tooth sensitivity. Remember mouthwash cannot replace toothbrushing and flossing or interdental brush. Use mouthwash in addition to daily cleaning routine may offer additional benefit in terms of plaque and gingivitis reduction. For those who have just undergone oral surgical procedures, toothbrushing may be hindered temporarily by the surgery. They should use of mouthwash.

### **CHEWING SUGAR-FREE GUM**

Chewing sugar-free gum can neutralise plaque acids and protect against decay, but it can give a false sense of security. Avoiding sugar in the first place is the best policy.

**Table 14. Choice of dental hygiene means**

Type I embrasure <sup>1</sup>	Use regular floss
Type II embrasure <sup>2</sup>	1. Floss (superfloss) — best choice 2. Interdental brush — second 3. Wooden interdental cleaner — third
Type III embrasure <sup>3</sup>	1. Interdental brush 2. Wooden interdental cleaner 3. Floss (superfloss)
Type III embrasure with a slight open contact/diastema	Superfloss
Type III embrasure with a wide open contact/diastema	Single tuft brush
Class I, II, III furcation	1. Toothpick in holder (perio-aid) 2. Rubber or plastic interdental tip
Class IV furcation	1. Interdental brush



	2. Superfloss 3. Single tuft brush
Buccoverted/lingoverted teeth	Single tuft brush — brush vertically
Missing teeth with a wide open space Edentulous areas Distal surface of most posterior tooth	Single tuft brush — brush vertically
Lingually inclined mandibular teeth	Single tuft brush — brush vertically
Diastema or open contact	Superfloss
Gingival cleft	Toothpick in holder (perio-aid)
Under Pontic	Floss threaded with floss
Under Pontic that has a clinically visible, exposed space between the Pontic and gingival margin	1. Superfloss 2. Interdental brush if the space is very large between the Pontic and gingival margin
Interdental cleaning between Pontic and abutment in a type I embrasure	Floss threaded with floss
Interdental cleaning between Pontic and abutment in a type II embrasure	1. Superfloss 2. Small cylindrical interdental brush
Interdental cleaning between Pontic and abutment in a type III embrasure	1. Interdental brush 2. Superfloss
Interdental care for fixed orthodontics	1. No best choice 2. Floss threader with floss 3. Superfloss 4. Interdental brush
Around facial brackets	Single tuft brush
Around facial gingival margins	1. Oral irrigation (water pick) 2. Toothpick in holder (perio-aid) 3. Single tuft brush 4. Interdental brush 5. Superfloss 6. Wooden interdental cleaner

1. The interdental papilla fills the gingival embrasure (Type I embrasure).
2. There is slight to moderate recession of the interdental papilla (Type II embrasure).
3. There is extensive recession or complete loss of the interdental papilla (Type III embrasure).

### Question 6. Professional oral hygiene

Professional oral hygiene is a set of activities that includes the patient motivation and instruction on proper oral care and hygiene state control, and the removal of dental deposits by a dentist or hygienist using special tools.

Professional hygiene can be carried out from 2 to 4 times a year.

Professional hygiene includes two concepts: scaling (removal of supra- and subgingival calculus using short, strong movement towards oneself) and root planning (it is smoothing the surface of the tooth root using moderate to slight movements towards oneself).

### **Classification of instruments for dental deposits removal**

Classification of instruments for dental deposits removal:

1. Periodontal probes.
2. Sickle scalers.
3. Probes.
4. Curettes.
5. Excavators.
6. Files.
7. Ultrasonic instruments.
8. Cleaning and polishing instruments.

**Probes and periodontal probes** are used for detection of dental calculus, carious cavities, detection of gingival pockets, their depth and location removing granulations from the gingival pocket. They are divided into sickle scalers, which are heavy instruments for removal of supragingival dental calculus, and curettes, slim instruments, used for subgingival scaling, root planing and removal of granulations from the pocket.

**Nabers probe:**

- curved;
- blunt for furcation areas.

Furcation areas can also be detected with a straight probe.

**Excavators, chisels, files** help to remove dental calculus, tightly connected with the tooth, and necrotic cement. But the application of these instruments is limited compared to curettes.

**Ultrasonic instruments** are used for scaling and curettage.

**Instruments for cleaning and polishing** teeth are represented with rubber cups, brushes of various configuration, strips, polishing tapes, polishing systems, for instance «Air-polish».

For manual removal of dental deposits special instruments are used: scalers, curettes, excavators, hoes, chisels.

Hand instruments may be made of the following materials:

- metal;
- metal with diamond coating;
- plastic;
- teflon.

Almost all the instruments have a single constructive principle. All the instruments have the following parts: a handle, a shank and a blade

The *Handle* should be easy to grasp with fingers, so that the instrument can be directed without tension, and all movements strictly controlled. It shouldn't have to be too slim and too heavy. The handle should be perfectly centered and its design should prevent rotation and sliding out of the instrument during work. Handles can be single- and double-sided, different in relief and diameter.

The *shank* of the instrument may be flexible, moderately flexible, rigid and very rigid; in shape — straight, curved, contraangled (with double angles).

The *Blade* is the working part, the design of the working end indicates the use of the instrument and determines its classification. It has its face, back and two lateral surfaces. Between the face and lateral surfaces there are sharp cutting edges which perform the work.

According to the shape of the working end the instruments for scaling are divided into scalers and currettes. Scaler's blade is sharpened and has a triangular shape in the cross-section.

The face is positioned at a 90° angle relatively to the shank. The instrument has two equal lateral cutting surfaces, which makes it universal. But the application of this instrument is limited within the supragingival area because of its sharpness and possible risk of damaging the gingiva. This instrument is indicated for cleaning interdental spaces. Scalers have various shapes of the working end, including a wide range of instruments from simple ones, indicated for massive calculus removal, to thin graceful instruments for root planing. The working end of the currettes has a semi-round shape in the cross-section — a rounded back and a tip. Such shape allows removal of subgingival dental deposits without harming the gingiva.

Currettes are divided into universal ones and special Gracey currettes. Universal currettes have 2 equal cutting edges and are designed for the work on all dental surfaces. Sickle scalers (sickles) have two cutting surfaces and a sharp tip. The sickle is used primarily to remove supragingival calculus. Because of the design of this instrument, it is difficult to insert a large sickle blade under the gingiva without damaging the surrounding gingival tissues. Small, curved sickle scaler blades can be inserted under ledges of calculus a few mm below the gingiva. Sickle scalers are used with a pull stroke. It is important to note that sickle sealers with the same basic design can be obtained with different blade sizes and shank types to adapt to specific uses.

The «mini» modification of the sickle's working end may be indicated for removal of moderate dental deposits from interdental spaces, and in scrappy operations as well.

**Hoe Scalars.** Hoe scalars are used for the scaling of ledges or rings of calculus. The blade is bent at a 99-degree angle; the cutting edge is formed by the junction of the flattened terminal surface with the inner aspect of the blade. The cutting edge is beveled at 45 degrees. The blade is slightly bowed so that it can maintain contact at two points on a convex surface. The back of the blade is rounded, and the blade has a minimal thickness to permit access to the roots without interference from the adjacent tissues. The instrument has 4 types with various degrees of the blade curve, which allows removal of dental deposits from distal, mesial, lingual and buccal surfaces. Hoes may reach gingival pockets up till 3 mm deep.

**Files.** Files have a series of blades on the base. Their primary function is to fracture or crush tenacious calculus. Files can easily gouge and roughen root surfaces when used improperly. Therefore they are not suitable for fine scaling and root planing. Mini-bladed currettes are currently preferred for areas where files were once commonly used. Files are sometimes used for removing overhanging margins of dental restorations.

**Chisel Scalars.** The chisel scaler, designed for the proximal surfaces of teeth too closely spaced to permit the use of other scalars, is usually used in the anterior part of the mouth. It is a double-ended instrument with a curved shank at one end and a straight shank at the other; the blades are slightly curved and have a straight cutting edge beveled at 45 degrees. The chisel is inserted from the facial surface. The slight curve of the blade makes it possible to stabilize it against the proximal surface, whereas the cutting edge removes the calculus without damaging the tooth. The instrument is activated with a push motion while the side of the blade is held firmly against the root.

**Universal Currettes.** Universal currettes have two cutting edges and rounded tip that may be inserted in most areas of the dentition (either mesial, or distal surfaces without changing the instrument) by altering and adapting the finger rest, fulcrum, and hand position of the operator. The blade size and the angle and length of the shank may vary, but the face of the blade of every universal curette is at a 90-degree angle (perpendicular) to the lower shank when seen in cross section from the tip. The blade of the universal curette is curved in one direction from the head of the blade to the toe. Universal currettes are indicated for supragingival removal of deposits, especially in cervical area, and subgingival curettage as well.

Different types are designed for anterior or posterior teeth.

**Gracey Currettes** have been used in dental practice for more than 50 years. Gracey currettes are representative of the area-specific currettes, a set of several instruments designed and angled to adapt to specific anatomic areas of the dentition. The Gracey currettes also differ from the universal currettes in that the blade is not at a 90-degree angle to the lower shank. The

term offset blade is used to describe Gracey curettes, because they are angled approximately 60 to 70 degrees from the lower shank.

This unique angulation allows the blade to be inserted in the precise position necessary for subgingival scaling and root planning, provided that the lower shank is parallel with the long axis of the tooth surface being scaled. These curettes have marks on the handle, which indicates for which surface the instrument is designed.

Gracey curettes are available with either a «rigid» or a «finishing» type of shank. The rigid Gracey has a larger, stronger, and less flexible shank and blade than the standard finishing Gracey. The rigid shank makes it possible to remove moderate-to-heavy calculus without having to employ a separate set of heavy scalers such as sickles and hoes. Although some clinicians prefer enhanced tactile sensitivity that the flexible shank of the finishing.

Extended shank curettes such as the Hu-Friedy. After Five curettes are modifications of the standard Gracey curette design. The terminal shank is 3 mm longer, allowing extension into deeper periodontal pockets of 5 mm or more. Other features include a thinned blade for smoother subgingival insertion and reduced tissue distention and a large-diameter, tapered shank. Mini-bladed curettes such as the Hu-Friedy Mini Five curettes are modifications of the After Five curettes. They feature blades that are half the length of the After Five or standard Gracey curettes. The shorter blade allows easier insertion and adaptation in deep, narrow pockets; furcations; developmental grooves; line angles; and deep, tight, facial, lingual, or palatal pockets. In any area where root morphology or tight tissue prevents full insertion of the standard Gracey or After Five blade, the Mini Five curettes can be used with vertical strokes, with reduced tissue distention, and without tissue trauma.

Function - easier insertion and adaptation in any area where root morphology or tight tissue prevents full insertion of standard Gracey or After Five blade:

- deep, narrow pockets;
- furcations;
- developmental grooves;
- line angles;
- deep, tight pockets.

**Langer Curettes.** This set of three curettes combines the shank design of the standard Gracey #5–6, 11–12 and 13–14 curettes with a universal blade honed at 90 degrees rather than the offset blade of the Gracey curette. This combination of the Gracey and universal curette designs allows the advantages of the area-specific shank to be combined with the versatility of the universal curette blade. The Langer #5–6 curette adapts to the mesials

and distals of anterior teeth; the Langer #1–2 curette (Gracey #11–12 shank) adapts to the mesial and distal surfaces of mandibular posterior teeth; and the Langer #3–4 curette (Gracey #13–14 shank) adapts to the mesial and distal surfaces of maxillary posterior teeth. These instruments can be adapted to both the mesial and distal tooth surfaces without changing instruments.

Zone-specific curettes are developed for efficient work on the definite surface of the tooth. Curettes are designed for anterior and posterior teeth, palatal/lingual and vestibular surfaces. The shape of the working end perfectly corresponds to the anatomy of the crown. Besides efficient removal of dental deposits, they cause minimal trauma to soft tissues, which significantly improves healing.

Finishing curettes are used for root planing and removal of dental calculus in deep pockets. The cutting edge is 70° angled from the shank, which makes only one edge working and perfectly efficient.

**Dental excavators** are used for removal of massive dental deposits from buccal and oral surfaces of teeth, removal of dental calculus from hardly accessible sites and for cleaning of concave surfaces of tooth and furcation zone. They may be one-, two- and three-angled

### **ULTRASONIC AND SONIC INSTRUMENTS**

Ultrasonic and sonic instruments may be used for removing plaque, scaling, curetting and removing stains. The two types of ultrasonic units are magnetostrictive and piezoelectric. In both types, alternating electrical current generates oscillations in materials in the handpiece that cause the scaler tip to vibrate. Depending on the manufacturer, these ultrasonic vibrations at the tip of the instruments of both types range from 20 000 to 45 000 cycles/second (also referred to as Hertz (Hz)). In magnetostrictive units, the pattern of vibration of the tip is elliptical, which means that all sides of the tip are active and will work when adapted to the tooth.

In piezoelectric units, the pattern of vibration of the tip is linear, or back and forth, meaning that the two sides of the tip are the most active.

Sonic units consist of a handpiece that attaches to a compressed air line and uses a variety of specially designed tips. Vibrations at the sonic tip range from 2000 to 6500 cycles per second, which provides less power for calculus removal than ultrasonic units. Ultrasonic and sonic tips with different shapes are available for scaling, curetting, root planing, and debriding during periodontal surgery.

For many years, only large, bulky tips designed for supragingival removal of heavy calculus were available. In recent years, however, thinner, more delicate tips designed for subgingival debridement have become available. All tips are designed to operate in a wet field and have attached water outlets. The spray is directed at the end of the tip to dissipate the heat generated by the ultrasonic vibrations. Within the water droplets of this spray

mist are tiny vacuum bubbles that quickly collapse, releasing energy in a process known as cavitation. The cavitating water spray also serves to flush calculus, plaque, and debris dislodged by the vibrating tip from the pocket. Sonic units do not release heat the way ultrasonic units do, but they still have water for cooling and flushing away debris.

***Air polishing devices (pneumatic polishing).*** In the early 1980s, a specially designed handpiece was introduced that delivers an air-powered slurry of warm water and sodium bicarbonate; this instrument is called the Prophy-jet. This system is effective for the removal of extrinsic stains and soft deposits.

The slurry removes stains rapidly and efficiently by mechanical abrasion and provides warm water for rinsing and lavage. The flow rate of abrasive cleansing power can be adjusted to increase the amount of powder for heavier stain removal. The results of studies on the abrasive effect of the air-powder polishing device on cementum and dentin show that tooth substance can be lost. Damage to gingival tissue is transient and insignificant clinically, but amalgam restorations, composite resins, cements, and other nonmetallic materials can be roughened. Airpowder polishing can be used safely on titanium implant surfaces. Patients with medical histories of respiratory illnesses, hypertension, hemodialysis, sodium-restricted diets and those on medications affecting the electrolyte balance are not candidates for the use of the air-powder polishing device. Patients with infectious diseases should not be treated with this device because of the large quantity of aerosol created. A preprocedural rinse with 0,12 % chlorhexidine gluconate should be used to minimize the microbial content of the aerosol. High speed evacuation should also be used to eliminate as much of the aerosol as possible.

### **Cleansing and polishing instruments**

**Rubber Cups.** Rubber cups consist of a rubber shell with or without webbed configurations in the hollow interior. They are used in the angle handpiece.

A good cleansing and polishing paste that contains fluoride should be used and kept moist to minimize frictional heat as the cup revolves. Polishing pastes are available in fine, medium, or coarse grits and are packaged in small, convenient, single-use containers. Aggressive use of the rubber cup with any abrasive may remove the layer of cementum, which is thin in the cervical area.

**Bristle Brushes.** Bristle brushes are available in wheel and cup shapes. The brush is used in the handpiece with a polishing paste. Because the bristles are stiff, the use of the brush should be confined to the crown to avoid injuring the cementum and the gingiva.

**Dental Tape.** Dental tape with polishing paste is used for polishing proximal surfaces that are inaccessible to other polishing instruments. The tape is passed interproximally while being kept at a right angle to the long axis of the tooth and is activated with a firm labiolingual motion. Particular care is taken to avoid injury to the gingiva. The area should be cleansed with warm water to remove all remnants of paste

**Pastes** used in professional hygiene are similar in composition to pastes for individual oral care, but are more abrasive. Abrasive filler, pumice, silicate or alumina, dioxypastes are used in pastes for professional hygiene, they are close in composition to pastes for individual oral care, but have greater abrasiveness. There are pumice, silicate or alumina, silica, zircon. It is desirable that they contain preparations of fluorine and weak antiseptic agents. The company Septodont produces pastes Detartrine (abrasive-silica), Detartrine Z (abrasive powdered zircon and silica), Detartrine Fluoride (zirconium oxide and ionized fluorine) Pastes do not need to be kept wet to reduce the frictional heat that occurs when the cup rotates. Too much use of the calyx can irritate enamel or dentin, which is very thin in the cervical area of the tooth. For processing approximate surfaces that are not available for other tools, strips are used (dental tape). Work the tape in the vestibule-oral direction very carefully, so as not to damage the gums.

### **Tests to the topic**

#### **1. Ninfected dental plaque includes:**

- a. Pellicle.
- b. Dental plaque.
- c. Tartar.
- d. Cuticle.
- e. Plaque.

#### **2. According to the International Classification of Diseases to dental plaque include:**

- a. Supragingival tartar.
- b. Subgingival plaque.
- c. Food residues.
- d. Pigment deposits.
- e. Subgingival calculus.

#### **3. Dental plaque begins to form after his complete removal later:**

- a. 2 hours.
- b. 8 hours.
- c. 20-30 minutes.
- d. 12 hours.



- e. 15 minutes.

**4. The plaque consists of the following microorganisms:**

- a. Str. Mutans.
- b. Str. Salivarius.
- c. Lactobacilli.
- d. Actinomycetes.
- e. All the answers are correct.

**5. Dental plaque can be removed by:**

- a. Individual oral hygiene.
- b. Professional oral hygiene.
- c. Can not be deleted.
- d. There is no need to remove it.
- e. It is possible to remove only the chemical method.

**6. Methods of detection of dental plaque are:**

- a. Visual.
- b. Drying.
- c. Probing.
- d. Percussion.
- e. Palpation.

**7. Methods for the removal of tartar are:**

- a. Chemical.
- b. Mechanical.
- c. Physical.
- d. All answers are wrong.
- e. Combined.

**8. By origin, dental deposits are:**

- a. Biological.
- b. Physiological.
- c. Pathological.
- d. All answers are correct.

**9. Pellicle is formed as a result of:**

- a. Spontaneous deposition of proteins in the oral fluid on the enamel surface.
- b. Microorganism vital processes.
- c. Fermenting food carbohydrates.
- d. All answers are correct.

**10. Dental plaque is:**

- b. Dense structured formation located above the pellicle.
- c. Reduced epithelium of the enamel organ.
- d. Cell-free film on the enamel surface.
- e. All answers are correct.

## **LESSON 6. CLINICAL MANIFESTATIONS OF THE GUMS INFLAMMATION**

The questions to be studied for the learning of the topic:

1. Anatomical and histological structure of the gums. Signs and stage of inflammation.
2. Risk factors in the occurrence of gingivitis.
3. Relationship between oral hygiene and inflammation in periodontal tissue.
4. Clinical manifestations of gingivitis.
5. The definition of GI index and PMA, Schiller-Pisarev's test.
6. Difference between gingivitis and periodontitis.
7. Treatment and prevention of the gums inflammation.

### **Question 1. Anatomical and histological structure of the gums. Signs and stage of inflammation.**

Gum is the part of the oral mucosa surrounding the teeth. Outside gums verge-cheat with oral mucosa covering the alveolar bone of the jaw. This boundary has the form of a wavy line and a well-traced because of different colors. Gum covering the alveolar bone, has a bright red color, because it is lined by non-keratinizing epithelium through which blood is good shine suck-hole lamina propria. Gums covered stratum epithelium, has a pale shade. Inside the gum goes into gum edge zone of the hard palate, or floor of the mouth. Gum is divided into three parts: the attached free gingival and interdental papillae.

Sticky part of the gums tightly adherent to the periosteum of the alveolar processes of the jaws. The free part of the gums - the edge - free adjacent to the tooth surface and is separated from it only by a narrow slit (sulcus). It has a strong attachment to periosteum and has some mobility. The dividing line between the free-fastening and gum is gum groove running parallel to the gingival edge of a distance 0.5-1.5 mm and level roughly corresponds to the bottom of the gingival sulcus. Gingival interdental papillae are areas gums triangular filling gap between adjacent teeth. Gum in the sulcus has the highest permeability, is used for administration of drugs.

Gum is subjected to constant mechanical stress during chewing, and this explains the features of the structure of its epithelium and connective tissue.

Gum is lined with stratified squamous epithelium of the stratum thickness approximate, but 255 microns.

Lamina propria of the gingiva is presented papillary and reticular layers. The papillary layer is formed by loose connective tissue containing a large amount of blood from the vessels and nerve-fibers with numerous nerve endings. Reticulated layer pre-sented a layer of dense fibrous connective

tissue rich in collagen fibers, thick bundles which are firmly attached to the periosteum of the gingiva (attached gingiva). In the lamina propria also gums woven bundles of collagen fibers are firmly bonded with cement gum tooth. Glands and submucosa are absence of gum.

The border between the attached gingiva and the free pass at the enamel-cement border and 40% of adults with vestibular surface appears in the form of gum sulcus. Free and attached gingiva is a certain size.

Free gingiva covers the cervical surface and has a smooth surface. The width of the free gingival is 0.8-2.5 mm. The width of the attached gingiva is 1-9 mm, and with age, it may increase. The surface of the attached gingiva bumpy, uneven.

Gum between adjacent teeth called interdental. On the labial and gingival sides of the top of the gingival papillae are located, and between them are saddle recess referred to as "saddle". There are three areas of gingival epithelium.

Sulcus is a groove between the edge of the gums and the tooth surface. The depth of the gingival sulcus is usually 0,1-0,5 mm, but in some patients it reaches 2-3 mm The bottom of the sulcus is located on the neck of the tooth level. The transition of tooth enamel in cement. Gingival fluid is a physiological environment that normally fills the gingival sulcus, jutting out into the gum to a depth of 1-3 mm. Depending on its depth varies the amount of gingival fluid. During the day in the mouth comes cavity 0.5-2 ml of gingival fluid. The maxillary its quantity more than in the lower.

The composition of gingival fluid are water, white blood cells, microorganisms, enzymes, proteins, exfoliated epithelium, immunoglobulins, however it has antimicrobial action. When clinically healthy gingiva, gingival fluid can be detected or not it appears in small quantities. With increasing intensity of gingivitis amount of fluid in the gingival sulcus is significantly increased.

Gingival fluid takes part in a mechanical furrow-washing and is nutritious environment for subgingival plaque microorganisms.

Inflammation is a soft tissue cellular and vascular response to local injury of physical, thermal, chemical, or microbial origin. Inflammatory periodontal diseases are no exception to this paradigm as local periodontal etiologic factors may be physical (factitial habits such as toothbrush abrasion or occlusal trauma), thermal, chemical (epithelial disorders associated with some mouth\ivashes, smokeless tobacco, aspirin, and cocaine), and microbial (dental plaque induced gingival diseases). The most common inflammatory periodontal diseases are caused by a local accumulation of bacteria.

Etiology is "the study or theory of the causation of any disease; the sum of knowledge regarding causes." Therefore, etiology is a noun that defines

the science of disease causation, but in common usage, etiology is a cluster of factors that contribute to disease (ie, the etiology of periodontal diseases).

Inflammation always starts with cell damage and death, and it ends with recovery-damaged tissue by proliferation. Depending on the nature of the dominant local process (alteration, exudation, proliferation) are three types of inflammation:

- 1) alterative - dominated necrosis, degeneration and damage.
- 2) exudative - characterized by impaired blood flow to the phenomena of exudation and leukocyte migration. By the nature of fluid inflammation may be serous, purulent, hemorrhagic, catarrhal (if a lot of mucus exudate).
- 3) proliferative - dominated by cell proliferation or hematogenous histogens pro-origin in the inflammation and cell infiltrates occur.

### **Question 2. Risk factors in the occurrence of gingivitis.**

Gingivitis is a common and mild form of gum disease (periodontal disease) that causes irritation, redness and swelling (inflammation) of gums. Because gingivitis can be mild, you may not be aware that you have the condition. But it's important to take gingivitis seriously and treat it promptly. Gingivitis can lead to much more serious gum disease (periodontitis) and eventual tooth loss.

Inflammation of the gums can be caused by a number of general and local factors.

#### **1. Local factors:**

1.1 - microorganisms in the plaque is the main risk factor for sore-of gums. Studies show that 80 - 90% of periodontal disease caused by activity of microorganisms of dental plaque;

1.2 - local exposure to chemicals (acids, alkalis, metal salts);

1.3 - exposure to high and low temperatures, radiation energy;

1.4 - infringement of functional load on periodontal (with malocclusion, edentulous, dysfunction of the temporomandibular joint, oral parafunctions and bad habits, carious tooth decay, excessive occlusion prosthesis or seal, and so on);

1.5 - Pathology architectonic vestibule of mouth: improper attachment of the ultrasonic bridle, buccal bands, small vestibule of the oral cavity.

1.6 - retention factors (orthodontic and orthopedic structures, overhanging edges of fillings and so on);

1.7 - chronic trauma (smoking, mechanical injury due to improper wire-my technique oral hygiene).

1.8 - Violation amount and composition of the oral fluid, which is normally a natural protective factor in the oral cavity.

## **2. Common factors:**

2.1 - chronic somatic diseases (for example, diabetes, cardiovascular disease, and so on);

2.2 - stress;

2.3 - malnutrition;

2.4 - taking certain medications;

2.5 - hereditary factor (the individual characteristics of the protective potential of the human organism);

2.6 - environmental factors (socio-economic, environmental conditions). Considered, for example, that insufficient cultural level of the person causes his dismissive attitude toward oral hygiene and thus creates a blab enabling environment for the development of periodontal disease.

2.7- occupational hazards.

### **Question 3. Relationship between oral hygiene and inflammation in periodontal tissue.**

The most common cause of gingivitis is poor oral hygiene that encourages plaque to form. Plaque is an invisible, sticky film composed mainly of bacteria. Plaque forms on teeth when starches and sugars in food interact with bacteria normally found in mouth. Brushing teeth at least twice a day and flossing each day removes plaque. Plaque requires daily removal because it re-forms quickly, usually within 24 hours.

Plaque that stays on teeth longer than two or three days can harden under gum line into tartar (calculus). Tartar can also develop resulting from the mineral content in saliva. Tartar makes plaque more difficult to remove and creates a protective shield for bacteria. usually can't get rid of tartar by brushing and flossing - need a professional dental cleaning to remove it.

The longer that plaque and tartar remain on teeth, the more they irritate the gingiva, the part of gum around the base of teeth. In time, gums become swollen and bleed easily. Tooth decay (dental caries) also may result.

### **Question 4. Clinical manifestations of gingivitis.**

Healthy gums are firm and pale pink. If gums are puffy, dusky red and bleed easily, may have gingivitis. Because gingivitis is seldom painful, can have gingivitis without even knowing it.

Signs and symptoms of gingivitis include:

- Swollen gums
- Soft, puffy gums
- Receding gums
- Occasionally, tender gums
- Gums that bleed easily when you brush or floss, sometimes seen as redness or pinkness on brush or floss

- A change in the color of gums from a healthy pink to dusky red
- Bad breath

Most dentists recommend regular checkups to identify gingivitis, cavities (caries) and other dental conditions before they cause troubling symptoms and lead to more serious problems. If notice any signs and symptoms of gingivitis, schedule an appointment with dentist. The sooner seek care, the better chances of reversing damage from gingivitis and preventing its progression to periodontitis.

**Complications.** Untreated gingivitis can progress to gum disease that spreads to underlying tissue and bone (periodontitis), a much more serious condition that can lead to tooth loss.

Periodontitis and poor oral health in general may also affect overall health. It's not completely understood - and researchers haven't established whether periodontal disease actually causes any of these conditions - but having periodontitis may be associated with:

- ✓ Heart attack
- ✓ Stroke
- ✓ Lung disease
- ✓ Premature birth or having a baby with low birth weight, in women

#### **Question 5. The definition of GI and PMA index, Schiller-Pisarev's test**

Dentists usually diagnose gingivitis based on symptoms and an examination of teeth, gums, mouth and tongue. Dentist will look for plaque and tartar buildup on teeth and check gums for redness, puffiness and easy bleeding. If it's not clear what has caused gingivitis, dentist may recommend that you get a medical evaluation to check for underlying health conditions.

**GI index** . The index was discussed in topic 2.

**Papillary-Marginal-Attachment Index.** PMA index is probably the oldest reversible index which was developed by Schour I and Massler M (1944). It was used to assess the extent of gingival changes in large groups for epidemiological studies. It was based on the concept that the extent of inflammation serves as an indicator of the severity of the condition. The presence or absence of inflammation is recorded in three areas of gingiva around the teeth.

*Selection of Teeth and Surfaces.* Three gingival units are examined for each tooth P = Papillary portion between the teeth

Papilla is numbered by the tooth just distal to it. Papilla is not present when teeth are separated by a diastema or there is an edentulous area.

Inflammation usually begins within the papilla at the col area.

Papillary changes - Mild gingivitis. M = Marginal collar around the teeth

It is located between papillae, attached by junctional epithelium, and demarcated from attached gingiva by the free gingival groove.

Papillary and marginal gingival inflammation - Moderate gingivitis.

A = Attached gingiva overlying the alveolar bone

Stippled gingiva between the free gingival groove and the mucogingival junction.

Spread of inflammation from papillary and marginal gingivitis into the attached gingiva - Severe gingivitis.

*Method.* All the teeth can be assessed starting from maxillary second molar of one side to the second molar of the other side and then mandibular second molar of the same side to the second molar of the other side. Third molars are not included. Adequate light and mouth mirror are used. Probe usually a blunt probe is used for pressing on gingiva.

**Table 15. PMA Scoring Criteria**

Papillary = P	Marginal = M	Attached = A
0 = Normal, no inflammation.	0 = Normal, no inflammation visible.	0 = Normal; pale rose, stippled.
1+ = Mild papillary engorgement, slight increase in size.	1+ = Engorgement, slight increase in size, no bleeding.	1+ = Slight engorgement with loss of stippling, change in color may or may not be present.
2+ = Obvious increase in size of gingival papilla, bleeding on pressure.	2+ = Obvious engorgement, bleeding upon pressure.	2+ = Obvious engorgement of attached gingiva with marked increase in redness, pocket formation present.
3+ = Excessive increase in size with spontaneous bleeding.	3+ = Swollen collar, spontaneous bleeding, beginning infiltration into attached gingiva.	3+ = Advanced periodontitis, deep pockets evident.
4+ = Necrotic papilla.	4+ = Necrotic gingivitis.	
5+ = Atrophy and loss of papilla (through inflammation).	5+ = Recession of the free marginal gingiva below the cemento-enamel junction as a result of inflammatory changes.	

*Scoring.* P-M-A for individual: count the number of P, M and A units scored and record separately as: P-M-A =?-?-? Keeping the total separate, as on adding the sum will not represent the area of the gingiva where the inflammation is present.

P-M-A for a group: The average of the P, M and A is computed by totalling each for all individuals and then dividing each number of individuals examined.



*Interpretation:*

- ✓ up to 25% - mild gingivitis;
- ✓ 25-50% - moderate gingivitis;
- ✓ 50% - severe gingivitis.

**Schiller-Pisarev's test (1963)**

The mucous membrane of the oral cavity is treated by a solution of potassium iodide for determination the intensity of the inflammatory process. Staining occurs in areas with deep connective tissue damage. It is associated with the accumulation of large amounts of glycogen at the places of inflammation. The color intensity and its area decrease in the case of remission of the inflammatory process or its termination.

*Technique:* the gum is treated by the solution of potassium iodide. The degree of staining is determined and areas of intense darkening of the gums are recorded in the examination card. The test can be evaluated in scores. The staining of the papillae is 2 scores, the staining of the gingival margin is 4 scores and the staining of the alveolar gums is 8 scores. Then, the total score is divided by the number of teeth in the area of which the study was carried out (usually 6) according to the formula:

$$\text{Iodine number} = \frac{\text{Sum of scores for each tooth}}{\text{The number of examined teeth}}$$

**Interpretation:**

Mild inflammation - up to 2,3;

Moderate inflammation – 2,67-5,0;

Severe inflammation – 5,33-8,0.

**Question 6. Difference Between Gingivitis and Periodontitis**

Gingivitis (gum inflammation) usually precedes periodontitis (gum disease). However, it is important to know that not all gingivitis progresses to periodontitis.

In the early stage of gingivitis, bacteria in plaque build up, causing the gums to become inflamed and to easily bleed during tooth brushing. Although the gums may be irritated, the teeth are still firmly planted in their sockets. No irreversible bone or other tissue damage has occurred at this stage.

### **Question 7. Treatment and prevention of the gums inflammation.**

Prompt treatment usually reverses symptoms of gingivitis and prevents its progression to more serious gum disease and tooth loss. Effective treatment requires professional care followed by stepped up oral hygiene at home.

Professional gingivitis care includes:

1. An initial evaluation and thorough dental cleaning to remove all traces of plaque and tartar
2. Instruction on effective home brushing and flossing techniques
3. Regular professional checkups and cleaning
4. Possibly fixing crowns or fillings (dental restorations) that make good hygiene difficultinitial professional cleaning will include use of dental instruments to remove all traces of plaque and tartar - a procedure known as scaling. Scaling may be uncomfortable, especially if gums are already sensitive or you have extensive plaque and tartar buildup.

Misaligned teeth or poorly fitting crowns, bridges or other dental restorations may irritate gums and make it harder to remove plaque during daily home care. If problems with teeth or dental restorations contribute to gingivitis, dentist may recommend fixing these problems.

Gingivitis usually clears up after a thorough professional cleaning - as long as you continue good oral hygiene at home. dentist will help you plan an effective at-home program.

Steps can take at home to prevent and reverse gingivitis include:

1. Get regular professional dental cleanings, on a schedule recommended by dentist.
2. Use a soft toothbrush and replace it at least every three to four months.
3. Consider using an electric toothbrush, which may be more effective at removing plaque and tartar.
4. Brush teeth twice a day, or better yet, after every meal or snack.
5. Floss at least once a day.
6. Use an antiseptic mouthwash, if recommended by dentist.
7. Use an interdental cleaner, such as a dental pick or dental stick specially designed to clean between teeth.

If you're consistent with home hygiene, you should see the return of pink, healthy gum tissue within days or weeks. Need to practice good oral hygiene for life, however, so gum problems don't return.

**Prevention.** The best way to prevent gingivitis is a program of good dental hygiene, one that begin early and practice consistently throughout life. A complete cleaning with a toothbrush and floss should take three to five minutes. Flossing before brush allows to clean away the loosened food particles and bacteria.

See dentist or dental hygienist regularly for professional cleanings, usually every six to 12 months. If have risk factors that increase chance of developing gingivitis, may need professional dental cleanings more often.

### **Tests to the topic**

#### **1. Indicate the key to determine the teeth gingival index GI:**

- a. 16,17,11,26,36,31,46,47.
- b. 16,21,24,36,41,44.
- c. Consider all the teeth.
- d. 22, 23, 24, 13,16.
- e. 11, 45, 33, 44, 16, 27.

#### **2. What shape is the apex of the interdental papillae in the anterior teeth is normal?**

- a. Round.
- b. Spherical.
- c. Crater.
- d. Peaked.
- e. Truncated.

#### **3. Indicate the clinical signs of inflammation of the gums:**

- a. A color change, the presence of periodontal pockets, tooth mobility.
- b. A change of color, texture, contour the gingival margin, surface bleed-ness.
- c. Changing the consistency, bleeding, increased number of gingival crevicular fluid.
- d. Availability of gums, discoloration of the gums.
- e. Changing the consistency of the gums, teeth tenderness to percussion.

#### **4. Select a color gum in chronic inflammation:**

- a. Coral-pink.
- b. A bright red.
- c. Dark red with cyanosis.
- d. Pink.
- e. Brown.

#### **5. Assess presence of bleeding gums is necessary to:**

- a. In the time of sensing.
- b. Within 30-40 seconds after probing.
- c. 1-2 seconds after sensing.
- d. 20 seconds after the probe.
- e. 10 seconds after the probe.

**6. Histologically, the gum comprises:**

- a. Stratified epithelium, lamina propria, the submucosa.
- b. Stratified epithelium, basement membrane, submucosa, minor salivary glands.
- c. Stratified epithelium, lamina propria.
- d. The lamina propria of the mucosa, submucosa.
- e. Minor salivary glands; submucosa.

**7. Indicate the risk factors for gum inflammation:**

- a. Microorganisms of dental plaque.
- b. Local traumatic factors.
- c. General somatic diseases.
- d. All of the above.

**8. Indicate the type of gums in case of acute inflammation:**

- a. A. Smooth, shiny, bright red, bleeds easily when touched, the contour is smoothed.
- b. Pink, dense, no bleeding.

**9. The surface of the normal gums is:**

- a. Smooth, shiny.
- b. Uneven, with evenly spaced elevations.

**10. Gum surface during inflammation is**

- a. Smooth, shiny.
- b. Uneven, with evenly spaced elevations.

## **LESSON 7. METHODS OF INVESTIGATION CHARACTERIZED DESTRUCTIVE PROCESS OF PERIODONTAL TISSUES**

1. Etiologic factors in periodontal diseases.
2. Clinical research methods characterized destructive processes in periodontal tissues.
3. The detailed study of periodontal tissue.
4. Periodontal indices.
5. X-ray diagnostics of periodontal diseases.

### **Question 1. Etiologic factors in periodontal diseases**

The microbiologic etiologic factor in periodontal diseases is dental plaque with dental calculus as probably the most significant local contributing factor. Food debris and the bacteria it contains is probably a major etiologic factor in root caries.

There is little dispute over the concept that bacteria are the primary etiologic factors in inflammatory periodontal diseases. In 1965, Loe and co-workers published their classic work that demonstrated that gingival health could be reliably achieved with immaculate oral hygiene and that gingival inflammation could be caused by the accumulation of plaque on the teeth. Light microscopic examination of tooth scrapings revealed that plaque was an adherent mat of bacteria, epithelial cells, and leukocytes encased in an amorphous protein and polysaccharide matrix, and that cocci, filamentous bacteria, spirochetes, and vibrios accumulated on teeth in an ordered sequence. The knowledge produced in this and later studies of plaque morphology and microbiology emphasized that plaque was a heterogeneous community of bacteria.

Generally speaking, bacteria associated with periodontal health are characterized as Gram-positive, nonmotile facultative anaerobes. Bacteria associated with disease are generally Gram-negative, motile, strictly anaerobic species. The cell wall of Gram-negative bacteria consists of a lipopolysaccharide base, also known as endotoxin, that has significant pathogenic potential. While over 350 distinct species of bacteria have been isolated from the oral cavity, relatively few are associated with gingival or periodontal inflammation. The list of strongly associated pathogenic bacteria includes:

- *Actinobacillus actinomycetemcomitans*
- *Bacteroides forsythus*
- *Fusobacterium nucleatum*
- *Peptostreptococcus micros*
- *Porphyromonas gingivalis*
- *Prevotella intermedia/nigrescens*

***What is Meant by the Term "Biofilm"?*** Biofilms form on inert surfaces where bacteria to bacteria cohesive interactions or bacteria to surface adhesive interactions are allowed to occur. Biofilms are heterogeneous composites of bacterial communities within a nonbacterial protein, polysaccharide, and glycoprotein matrix of bacterial and salivary origin. The matrix allows for a "circulation" of nutrients and bacterial metabolites between communities and the environment outside the biofilm. There are extreme variations in oxygen levels ranging from highly aerobic areas within fluid channels to almost completely anaerobic areas in microcolonies.

***Nonspecific Plaque Hypothesis.*** The basic tenets of the nonspecific plaque hypothesis state that inflammatory periodontal diseases (and possibly caries) are caused by composite effect of bacterial colonization and maturation on the surfaces of teeth, not by specific bacteria themselves. Gingival disease is the outcome from release of bacterial metabolites (such as butyrate or other short chain fatty acids) and immunogenic bacterial antigen components, such as lipopolysaccharide (endotoxin) from Gram-negative cell walls during plaque growth. Inflammatory disease is the outcome of a microbial mass that is in excess of the local defense mechanisms of the host.

***Specific Plaque Hypothesis.*** The specific plaque hypothesis states that periodontitis is an infection caused by a limited number of periodontal microorganisms, and that these microorganisms characterize the plaque biofilms associated with periodontitis but not gingivitis or gingival health. It appears that of the 300+ identifiable species found in the oral cavity, only a small proportion (10-12 species) are actually found in active periodontitis sites. The bacteria believed to be pathogens in periodontitis do not conform to the classic dogma for microbial pathogenicity (ie, Koch's Postulates). The current understanding of mixed infections, bacterial invasion, virulence factors, conducive bacteria habitats, the role of so-called "beneficial species", and the susceptibility of the host have rendered Koch's Postulates obsolete when it comes to periodontitis.

***Supposed Periodontal Pathogen.*** The criteria for implicating oral microorganisms as periodontal pathogens are:

1. The microorganism must be associated in high numbers in active periodontitis lesions and either absent (not cultivable) or in low numbers in gingivitis or healthy sites. The numbers of the microorganism should have increased to a threshold level before the onset of disease.
2. The elimination of the microorganism, or its numerical reduction below threshold levels, should parallel remission of active disease.
3. There should be a specific host immune response against the organism (ie, elevated serum, salivary, and crevicular fluid antibody titers).
4. The microorganism should evoke virulence factors that contribute to its pathogenicity or explain disease pathobiology.

5. The microorganism should produce periodontitis in animal model systems.

**Features of Periodontal Pathogens** certain features in common. Most of them have a Gram-negative cell wall. The outer membrane of the Gram-negative cell wall contain lipopolysaccharide (LPS) which has endotoxin activity. Typically, LPS containing Gram-negative cell wall extracts are capable of promoting bone resorption, inhibiting osteogenesis, chemotaxis of neutrophils, and other events associated with active periodontitis. Some pathogens release a LPS that suppresses the innate immune response. Many periodontal pathogens are strict or facultative anaerobes and are asaccharolytic, permitting survival in the restricted ecosystem of the periodontal pocket. Among the strict anaerobes is the only presumptive periodontal pathogen with a Gram-positive cell wall, *Peptostreptococcus micros*.

*Actinobacillus actinomycetemcomitans* and *Porphyromonas gingivalis* are the best studied and have been designated, along with *Bacteroides forsythus*, as true etiologic agents in periodontitis because of the host of virulence they produce and their ability to invade gingival tissues. *Prevotella intermedia/nigrescens* and *Fusobacterium nucleatum* have been widely studied as well, and appear to satisfy all criteria for periodontal pathogenicity. Because *P. intermedia* and strains of *F. nucleatum* have also been found in areas of severe gingival inflammation without evidence of attachment loss, controversy exists as to their true periodontal pathogenicity.

*Campylobacter rectus*, *Eikenella corrodens*, *Eubacterium* species, *Selenomonas* species, enteric rods/*Pseudomonas* species, and *Treponema* species satisfy some, but not all, criteria with any degree of confidence. Nonetheless, they remain among the list of periodontal pathogens, and microbiology testing services commonly report their presence among cultivable flora. Relative risk values of periodontal pathogens in periodontal sites have emerged from archival reviews of data bases located in commercial testing facilities. The relative risk of a microorganism as a pathogen is often expressed as percent of total cultivable bacteria in a given culture. For example, the cultivability of *A. actinomycetemcomitans* at levels at or above 0.01% indicates a periodontal site at risk for active disease. The risk for *P. gingivalis*, *C. rectus*, *P. intermedia*, and *P. micros* in periodontal sites is 0.1%, 2%, 2.5%, and 3%, respectively.

**Risk Factors for Periodontal Diseases.** The expression risk in this context means that, in the presence of a given factor, injury to or loss of periodontal tissue is a possibility. Risk factors may be local or systemic in nature. Local contributing factors to the etiology of periodontal diseases fall into two general categories: *anatomic or iatrogenic*. They share in common their ability to either facilitate bacterial plaque, and therefore calculus,

accumulation/retention or their ability to interfere with plaque/calculus removal.

**Table 16. The local anatomic risk factors.**

<i>Risk factor</i>	<i>Its characteristic</i>
<b>1. Furcation anatomy.</b>	In many instances, the entrance of bifurcations or trifurcations is restricted enough to limit access for mechanical root instrumentation. Once access to the intrafurcal space has been achieved, concavities in the furcal aspects of molar roots will limit instrumentation as well.
<b>2. Intermediate bifurcation ridges</b>	extending from the mesial furcation surface of the distal root across the roof of the bifurcation to the distal surface of the mesial root of mandibular molars. These common anatomic deformities interfere with a patient's ability to effectively remove plaque biofilm.
<b>3. Cervical enamel projections (CEP).</b>	CEPs are tooth developmental deformities of the CEJ found on molars. They are classified according to their involvement in tooth furcations. A Grade I CEP presents with minimal projection of enamel toward the entrance of the furcation. A Grade II CEPs approximates the entrance of the furcation, and the tip of a Grade III CEP is well within the furcation.
<b>4. Palato-gingival grooves (PGG).</b>	PGGs are tooth developmental deformities of maxillary central and lateral incisors. They begin in lingual pits and extend vertically onto root surfaces. PGGs could, on rare occasions, extend to the root apex. PGGs are commonly associated with increased gingival inflammation, plaque accumulation, and probing depth.
<b>5. Open contacts and food impaction.</b>	Open contacts between teeth may be anatomical in origin, iatrogenic in origin, or be due to caries and pathologic migration of periodontally involved teeth. Food impaction is defined as the forceful wedging of food between teeth. Any other accumulation of food or food debris around teeth should be categorized as food retention and is probably less threatening to the periodontium. Food impaction and subsequent retention may contribute to root caries in individuals who do not



	perform proper oral hygiene interdentally. Open contacts by themselves probably do not contribute to periodontal pathology, but, in the presence of food impaction, open contacts have been associated with periodontal destruction. This may be particularly noticeable in periodontitis cases where the progress of disease is in its early stages or particularly obvious where periodontitis is isolated to sites of open contacts/food impaction.
<b>6. Other anatomic risk factors of potential etiologic importance</b>	The width of the space between teeth and root proximity (so-called "kissing roots").

**Table 17. The iatrogenic risk factors.**

<i>Risk factor</i>	<i>Its characteristic</i>
<b>1. Overhanging dental restorations.</b>	Since dental restorations remain the mainstay of dental practice, it is not surprising that overhanging dental restorations are arguably the most common form of iatrogeny to affect marginal periodontal health. Overhanging and improperly placed dental restorations can be physically irritating, be plaque retentive, foster the growth of periodontal pathogens, alter the morphology of the interdental space, and violate the dentogingival junction (see 2 below). By virtue of their roughness and overall bulk, they may also interfere with interdental plaque control.
<b>2. Violation of the "biologic width".</b>	After overhanging restorations, iatrogenic invasion of the biologic width may be the next more serious insult to the periodontium a dentist can make. The impact of this insult is usually permanent as the margins of dental restorations are inevitably placed in the wake of the insult. The biologic width is one of nature's constant dimensions. It is most constant within individuals and less constant between individuals. If it is injured, it will repair. If however, restorative materials render the invasion of the biologic width permanent, periodontitis will produce apical migration of the junctional epithelium, resorption of crestal alveolar bone, loss of periodontal attachment, and possible vertical

	osseous defects. A new biologic width will repair a few mms apical to its original position on the tooth. This represents a net loss of attachment on the tooth.
<b>3. Open contacts and food impaction related to inadequate restorative dentistry.</b>	The impact of food impaction through open contacts created by iatrogeny offers the same threat to the periodontium as food impaction associated with open contacts that have resulted from growth and development or occlusal wear.
<b>4. Occlusal traumatism</b>	Associated with inadequate dentistry in 1, 2, and 3 above.
<b>5. Additional local iatrogenic risk factors for periodontal diseases</b>	Removable partial dentures and overdentures, fixed bridges, removal of third molar teeth in older adults, placement of fixed orthodontic appliances, and orthodontic movement of periodontally involved teeth.

**Systemic diseases and/or conditions that are contributing factors for periodontal diseases.** Aside from the medications that affect the clinical presentation of plaque-induced gingival diseases (nifedipine for control of hypertension, phenytoin for control of epileptic seizures, and cyclosporine to control organ transplant rejection), most systemic diseases and conditions that may affect periodontal diseases generally alter host barrier and host defense mechanisms. The impact of diminished host susceptibility, along with the diverse virulence mechanisms invading microorganisms possess, help to explain the individual variations in periodontal disease patterns we see in systemically ill periodontal patients. An assessment of systemic contributions to periodontal diseases in our patients is critical to periodontal diagnosis and/or treatment planning.

The systemic diseases and conditions that commonly affect periodontal diseases are: Uncontrolled type I and type II diabetes mellitus, HIV/AIDS, hormone imbalances, genetic predisposition, medications, smoking, and malnutrition.

**Table 18. The iatrogenic risk factors.**

<i>Systemic diseases and conditions</i>	<i>Its characteristic</i>
<b>1. Diabetes mellitus.</b>	The incidence of the disease seems to vary according to ethnic origin. DM is an aberration in carbohydrate, lipid, and protein metabolism. Most of the morbid complications of DM stem from long-term impaired glucose metabolism. The characteristic

	<p>hyperglycemia of uncontrolled DM is the basis for most of the vascular, cellular, and immune changes associated with the disease.</p> <p>Epidemiologic data has made clear associations between increased severity of periodontal diseases and uncontrolled type I and type II diabetes mellitus. Type I and type II uncontrolled diabetics tend to present with more gingival inflammation, more loss of periodontal attachment, and radiographic evidence of more bone loss than controlled or nondiabetic individuals. There is agreement that periodontal patients whose DM is well controlled may receive periodontal therapy without restrictions including periodontal surgery and implant placement. Uncontrolled diabetics, poorly controlled diabetics, or diabetics whose control is unknown should only receive emergency periodontal therapy, and that treatment should be performed with intraprocedural and/or postoperative antibiotic coverage. The patient's physician may also prescribe insulin or other antihyperglycemic agents to help limit post-operative infections or complications in wound healing.</p>
<b>2. HIV/AIDS.</b>	<p>Given the immunosuppressed state of these individuals (decreased CD4 lymphocytes), an expectation for severe periodontitis in patients with HIV/AIDS is reasonable. Indeed, these individuals suffer from other bacterial, viral, and fungal diseases more than those without HIV infection. Many succumb to these infections. Early studies of the periodontal status in AIDS patients indicated that these individuals showed increased severity of periodontal diseases. HIV-gingivitis (linear erythema) and HIV-periodontitis (necrotizing ulcerative periodontitis) categories of periodontal diseases were quickly proposed to designate the unique clinical characteristics of periodontal diseases in this group. Recently, the issue has been challenged by those who report no increases in the prevalence or extent of periodontal diseases among HIV-positive individuals.</p>
<b>3. Smoking.</b>	<p>Due to the vasoconstrictor effect of nicotine and the paralysis by carbon monoxide on the ability of</p>

	<p>hemoglobin to transport oxygen, it is understandable that smoking is a serious environmental risk factor for periodontal diseases. The length of time an individual has been smoking and the frequency of smoking play contributory roles in the severity of periodontal disease in smokers. Smokers also have a greater accumulation of plaque and calculus than nonsmokers and may be more at risk to harbor periodontal pathogens. While probing depth reduction following conventional nonsurgical and surgical periodontal therapy has been reported in smokers, the amount of reduction has been reported as less than that achieved in nonsmokers. A growing body of evidence suggests strongly that the failure rate of implant therapy is higher in patients who smoke. It is not uncommon for a therapist to recommend against the placement of dental implants in smokers. Patients must be counseled in this regard and supported in their attempts to overcome their addiction.</p>
<p><b>4. Sex hormone imbalances.</b></p>	<p>The most notable changes in the periodontium that are affected in part by hormonal changes occur in women in their childbearing years. In the case of pregnancy, progesterone and estrogen levels increase to levels that are several orders of magnitude greater than those seen during a normal menstrual cycle. Varying degrees of a reversible "pregnancy gingivitis" are common during pregnancy. The biologic impact of hormone changes range from the release of inflammatory mediators that increase vascular permeability (prostaglandins), the alterations in immunoregulation and pro-inflammatory regulators, the imbalances in the fibrinolytic system, and the abundant growth of the periodontal pathogen, <i>P. intermedia</i>. Because the duration of pregnancy is relatively short, hormonal changes associated with pregnancy have little effect on the more irreversible progress of periodontitis. Oral contraceptives mimic the hormonal levels seen during pregnancy, and it is not uncommon to find pregnancy-like changes in patients using birth control pills (BCP). Because gingival sex hormone concentrations tend to be lower</p>

	<p>during normal menstruation, it is not unexpected that women in their childbearing years may present with "cyclic" episodes of increased gingival inflammation. The most important concern of the dentist in managing patients who present with gingival disease related to hormone imbalances is to be certain that inflammatory disease control measures are effective. This is particularly important in women who are pregnant because data exists to suggest a relationship between periodontal infections (periodontitis) and preterm low birth weight babies. Antibiotics should be used only after a medical consultation in patients who are pregnant. Although controversial, there are reports of decreased effectiveness of oral contraceptives in individuals taking certain antibiotics. Individuals who are taking BCPs should be advised that the use of prescribed antibiotics such as tetracyclines and some penicillins may interfere with the action of BCPs. To avoid unwanted pregnancy, these individuals should be so warned and use alternative methods of birth control while taking antibiotics.</p>
<p><b><i>5. Genetic predisposition for periodontal diseases.</i></b></p>	<p>There is general agreement that individual responses to plaque bacteria vary. It has been suggested that disease pattern variations could be based, in part, on underlying genetically based differences in immune function. Indeed, the association of:</p> <ul style="list-style-type: none"> <li>a. Neutrophil receptor defects</li> <li>b. Antibody responses (IgG<sub>2</sub>) to periodontal pathogens</li> <li>c. certain histocompatibility antigens (HLA)</li> <li>d. Lymphocyte immune regulatory defects in patients with aggressive periodontitis adds credibility to this concept.</li> </ul> <p>Studies in twins indicate that many of the clinical variations seen in chronic periodontitis can be attributed to individual genetic differences. Recent reports of genetic pleomorphism in IL-1 genes and the elevated production of proinflammatory mediators, such as IL-1 , add another dimension to the impact genetic variations among individuals have on the patterns of chronic periodontitis.</p>

## **Question 2. Clinical research methods characterized destructive processes in periodontal tissues.**

The goals of the patient examination of with periodontal pathology are assessment of the general condition of the body, the clinical characteristics of periodontal disease, the identification of common and local etiological and pathogenetic factors, the definition of the form, stage and nature of the course disease.

Diagnosis of destructive processes in periodontal tissues should include:

- an assessment of the general condition of the patient,
- condition of the oral cavity,
- the detailed study of periodontal disease.

### **Survey plan:**

1. Anamnesis of life. Profession, the presence / absence of occupational hazards, the nutrition, the transferred diseases, stressful conditions, ecological, social, living conditions of life, hygiene of the mouth. Bad habits (in the past and / or the present time), whether the patient smokes (as much, for a long time). Predisposition to allergic reactions. Hereditary relation: do not suffer from similar diseases the next of relatives (parents, sisters, brothers). The patient should be examined and receive the conclusion of other specialists (therapist, endocrinologist, neurologist, allergist and other doctors) if necessary.
  2. Anamnesis of the disease. Complaints, the time of their appearance, the dynamics of development, possible causes of them; whether there were exacerbations, their reasons. Typical for periodontal disease are complaints of bleeding gums, the presence of dental deposits, itching and burning in the gums, bad breath, teeth mobility and displacement, the formation of three and diastema.
  3. Inspection. General appearance, facial expression, the presence of pathology in the visible skin, in the soft tissues of the maxillofacial region, the presence or absence of asymmetry; the condition of the lips, the corners of the mouth, diction, the degree of opening the mouth. A healthy person has a symmetrical face, lips are quite mobile, mouth opening, jaw movements are free, lymph nodes are not enlarged. Condition of function and parafunction of the maxillofacial region:
    - ✓ movements in the temporomandibular joint,
    - ✓ chewing,
    - ✓ oral habits (paving the tongue between the teeth while swallowing).
- 3.1. Inspection of the oral cavity.* During the inspection should follow a certain plan and follow the sequence.

**Table 19. Inspection of the oral cavity**

I. Smell from the mouth	4 groups of causes of bad breath: ENT diseases, diseases of the stomach, respiratory organs and oral cavity. Bad breath is caused by purulent discharge from the periodontal pocket or ulcerative necrotic gingivitis. The decay of food residues in the retention sites, desquamated epithelial cells and leukocytes increase the unpleasant smell. The unsatisfactory condition of oral hygiene, the presence of prostheses, carious teeth, butts, sinuses that occur in the case of chronic periodontitis make worse it.
II. Saliva	The amount, color, texture, viscosity, transparency, smell of saliva.
III. Lips	Inspect with open and closed mouth (color, shine, texture, moisture).
IV. Vestibule of the oral cavity	The depth, color of the mucous membrane, the intensity and tension of the frenulums, the place of their attachment on the alveolar process, the width of the attached gingiva. Determination the depth of the vestibule: measure the distance from the edge of the gum to its bottom with a graded instrument. The vestibule is considered small if its depth is not more than 5 mm, average - 8-10 mm, deep - more than 10 mm. Assessment of frenulums condition: a normal or simple frenulum is a thin triangular fold of mucous membrane with a wide base on the lip and ending in the middle line of the alveolar process approximately at a distance of 0.5 cm from the gingival margin. Anomalies of frenulums are characterized by the place of attachment, shape, size.
V. Oral mucosa	Examine the mucous of the cheeks from the corner of the mouth to the palatine tonsil (pigmentation, discoloration, etc.). The healthy mucous membrane of the mouth is pale pink or pink, does not bleed, adjoins tightly to the teeth.
VI. Tongue	Evaluate the consistency, mobility, all kinds of papillae.
VII. The bottom of the oral cavity	A change in color, vascular pattern, and relief is recorded.
VIII. Bite	Physiological types of bite are orthognathic, direct, progenic and biprognatic. <i>Orthognathic bite</i> is the front teeth overlap the lower and have an inclination forward in relation to the jaw. <i>Direct bite</i> is characterized by the

	<p>direct contact of the cutting surfaces of the front teeth and the clivus of the cutting tubers of the chewing teeth. The teeth and alveolar processes of the jaws are arranged vertically according to the shape of the jaws.</p> <p><i>Progenetic bite</i>: lower incisors overlap the upper ones. Incisors of the upper jaw touches the lingual surface of the incisors of the lower jaw. The alveolar part is located slightly in front of the body of the lower jaw. This type of physiological bite is a risk factor and aggravates the course of gingivitis and periodontitis (because the teeth are in permanent injury). <i>Biprognathic bite</i>: the front teeth of the upper and lower jaws are bent forward. The overlap of the upper and lower teeth between them is preserved. The periodont of the front teeth is in a condition of permanent injury.</p>
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#### IX. Examination of the dentition:

- 1) the number of removed teeth. The reason for their removal;
- 2) hypersensitivity of dentin;
- 3) the state of approximal contact of the teeth;
- 4) mobility;
- 5) percussion of teeth;
- 6) the presence of pathological migration;
- 7) occlusal contacts.

*IX.2. Hypersensitivity of dentin* occurs due to gingival recession, the formation of wedge-shaped defects, pathological abrasion due to traumatic occlusion, parafunction of the maxillofacial area (bruxism) during periodontal diseases. Diagnostic methods:

- 1) thermal stimuli (cold, heat).
- 2) tactile stimuli. The method of probing and its variations is used to identify tactile sensitivity: a) probing with a dental probe; b) probing with a special probe Yeaple with different pressure forces.
- 3) evaporative stimuli (air).

The wedge-shaped defect appears in the cervical area of the tooth in the form of a triangle, the apex of which is directed to the cavity of the tooth. In the defect there are two surfaces. One of them is almost horizontal located slightly above the gingival margin. The second surface makes an angle of almost 45° with the first, and its plane can partially extend (or continue) to the enamel in the cervical area of the tooth. The surface of the defect is smooth, polished to a shine, rarely with sharp edges. Wedge-shaped defects are localized mainly on the vestibular surface and less often on the lingual (palatal) and lateral.



Gingival recession is a progressive displacement of the gums in the apical direction. There are traumatic, symptomatic (one of the symptoms of gingivitis and periodontitis) and physiological (a sign of aging of the body and periodontal tissues) recession.

By localization:

- ✓ localized gingival recession occurs more often in 1-2 teeth.
- ✓ generalized is observed in a group of teeth (most common in periodontitis).
- ✓ systemic is observed in all teeth and is due to systemic factors.

*IX.3. The state of approximal contact of the teeth.* The degree of destruction of occlusal, proximal and cervical areas by caries, the presence and quality of restorations in the gum area significantly affects the results of subsequent periodontological interventions.

*IX.4. Mobility.* There are three degrees of tooth mobility: first - the tooth leans in the vestibular-oral (labial-lingual) direction within the width of the cutting edge (1-2 mm); second - except that indicated at the first degree of mobility, there is mobility in the medio-distal direction; third - in addition to these movements, the tooth is visually movable in the vertical direction.

*IX.5. Percussion of teeth.* Vertical, horizontal and comparative.

*IX.6. The presence of pathological migration.* The displacement of the teeth is one of the early and reliable signs of periodontal pathology. At the same time, gaps appear between the teeth, food gets into them during chewing, injuring the periodont. The most characteristic shift of the teeth is in the vestibular direction. The teeth that do not have nearby or absence antagonists move most intensively.

*IX.7. Occlusal contacts.* Occlusal relationships of the upper and lower dentitions are displayed on the occludogram. Its definition: a plate of pink tooth-technical wax is placed between the teeth, covered from the bottom surface with aluminum foil. Dentition closed in the position of the central occlusion under the supervision of a dentist. Teeth imprints remain on the wax. The deepest is in areas of premature contact. A special card is filled in periodontal practice. The dentist notes there the degree of pathological changes and the area of each tooth during the initial examination of the patient, the dynamics of the condition during treatment.

### **Question 3. The detailed study of periodontal tissue.**

1) The involvement of the gums in the pathological process. Normally, there is a free gum and an attached gum, the boundary between which is the so-called gingival groove running parallel to the edge of the gum at a distance of 0.5-1.5 mm. The *width* of the free gum is about 0.5-1.5 mm and is relatively constant in contrast to the attached gingiva, which varies between 1-9 mm. The width of the attached gingiva depends on the shape of the

alveolar process and the type of bite, the position of individual teeth. It is individual for each person.

*Color.* The gums pale during anemia, keratosis. It becomes bright red in cases of acute inflammation, polycythemia, and desquamation. Cyanosis is characteristic of chronic inflammatory processes, leukemia and diabetes. The intensity of the color of the gums changes during pathological changes. Gum color changes may occur in a group of teeth or be generalized, occurs only in the gingival papilla, or covers the gingival margin. There may be a diffuse discoloration of the gums, extending to the free and attached parts of it.

*Surface texture.* The surface of the attached gum normally has evenly spaced slight elevations. They make it look like an orange peel. The surface of the gums can become smooth, shiny during the development of pathological changes. This is observed in the case of edema and inflammation.

*The consistency of the gums* is determined by palpation. Normally, the gum is elastic. Loose, pasty or compacted is in case of pathological changes.

*Contour shape.* Normally, interdental nipples have a peaked shape. The gum thickens, the gingival margin acquires scallop in the case of chronic inflammatory processes. But there are other options for changing the contour.

*The size.* An increase in the size of the gums in the case of hypertrophic gingivitis leads to a change in the shape of the papillae, their tops are rounded and sometimes acquire a spherical shape. An increase in the size of the inflammatory gums is observed in the case of the edematous form of hypertrophic gingivitis, gingival and periodontal abscesses. These changes are localized and generalized. Chronic inflammatory hypertrophy can also spread to the gingival margin. Sometimes an increase in gum volume is non-inflammatory character and is associated with symptoms of hyperplasia. Such conditions occur during the treatment of patients with epilepsy with anticonvulsant drugs.

*Position.* Gingival recessions are observed during the determination of the location of the gingival margin in relation to the necks of the teeth. It is located below the enamel-cement border. Recessions can be generalized and localized. Localized gingival recessions are frequently on the vestibular surface of the lower jaw near the frontal teeth and on the palatal surface of the upper first molars. During the examination of the gums pay attention to the presence of periodontal fistula, scarring and morphological elements of the lesion.

2) Supragingival and subgingival calculus. Calculus (or tartar) is a form of hardened dental plaque. They are localized mainly in the cervical area of the teeth, retention areas, on the surfaces of the teeth adjacent to the excretory ducts of the salivary glands. The amount of calculus depends on the individual characteristics of the organism, the state of mineral metabolism, hygienic care of the oral cavity, the presence of an inflammatory process in

the periodontium and other conditions. Calculus is supragingival and subgingival. They differ in the mechanism of formation, localization, hardness and influence on the development of pathological processes in the periodont. *Supragingival calculus* is localized in the neck of the teeth. It is easily detected during the inspection, softer, well-stained with food colorants or special reagents. *Subgingival calculus* is located under the gum and is not detected during the inspection. It is firmly connected with the cement of the root of the tooth, painted in dark color with blood pigments.

3) The presence of bleeding gums. The symptom of bleeding gums is evaluated during an easy probing of the gingival groove, within 30 seconds after probing.

4) Presence of periodontal pocket. The pocket is a deepening of the physiological gingival groove caused by the destruction of the periodontal attachment and proliferation of the epithelium in the apical direction. Signs:

1. Enlarged, bluish-red marginal gingiva with a 'rolled' edge separated from the tooth surface.
2. A bluish-red vertical zone extending from the gingival margin to the alveolar mucosa.
2. A break in the faciolingual continuity of the interdental gingiva.
3. Shiny, discolored and puffy gingiva associated with exposed root surfaces.
2. Gingival bleeding, purulent exudate from the gingival margin.
3. Mobility, extrusion and migration of teeth.
4. The development of diastema where none had existed previously.

Types of pockets:

- A. False (gingival).
- B. Periodontal (true) pocket.
- C. Combined pocket.

Depending upon the number of surfaces involved:

- a. Simple pocket: involving one tooth surface.
- b. Compound pocket: involving two or more tooth surfaces.
- c. Complex pocket: where the base of the pocket is not in direct communication with the gingival margin. It is also known as spiral pocket.

5) The involvement of furcation in the pathological process. The severity degree of furcation:

Degree-1. Disappearance of tooth attachment in the area of roots furcation. Periodontal pocket in the area of roots furcation with a depth of less than 3 mm. Radiological changes are not present.

Degree-2. The disappearance of the attachment of the tooth in the area of roots furcation, which advanced under the crown of the tooth, but on the opposite side of the alveolar wall in the area of furcation remained

untouched. The depth of the periodontal pocket is more than 3 mm. The probe does not pass through in the area of furcation. Deep curved pocket or horizontal pocket in the furcation area. Small or middle area of enlightenment between the roots on the radiograph.

Degree-3. The disappearance of attachment in the area of furcation, which advanced under the crown of the tooth with the formation of a tunnel, the upper wall of which is the crown of the tooth. The entrance to the tunnel is covered by a gum. Periodontal probe passes through the tunnel. Radiographically visible extensive area of enlightenment in the inter-root area.

Degree-4. The same lesion as with degrees-3. But the loss of the gums is expressed so that the tunnel under the tooth is visible by the eye.

6) Trauma from occlusion. Traumatic occlusion is observed if one tooth or group of teeth experiences a traumatic load during the closing.

#### **Question 4. Periodontal indices**

**CPI index (P.A. Leus) and Community Periodontal Index of Treatment Needs (CPITN)** were discussed previously in topics 2 and 5.

**Periodontal Index (PI).** Russell [1956] developed an index for measuring periodontal disease that could be used in population surveys. It can be based solely upon the clinical examination, or it can make use of dental X-rays if they are available. It places greater emphasis on advanced disease. PI determines the periodontal disease status of populations in epidemiologic studies. Each tooth is scored according to the condition of the surrounding tissues. On examination, each tooth is assigned a score using the following criteria:

**Scoring.** Each tooth is scored separately according to the following criteria. Rule: When in doubt, assign the lower score.

#### ***Reasons for its Widespread Use***

- Ease of use
- Clarity of criteria
- Reasonable comparability of results

**Table 20. Scoring Criteria of Russell's Periodontal Index**

<i>Criteria for field studies</i>	<i>Additional X-ray criteria</i>	<i>Score</i>
Negative (neither overt inflammation in the investing tissues, nor loss of function due to destruction of supporting tissues)	Radiographic appearance is essentially normal	0
Mild gingivitis (overt area of inflammation in the free gingivae, but this area does not circumscribe the		1

tooth)		
Gingivitis (inflammation completely circumscribes the tooth, but there is no apparent break in the epithelial attachment) (Not used in field study)		2
Used only when radiographs available	Early, notchlike resorption of the alveolar crest	4
Gingivitis with pocket formation (the epithelial attachment is broken, and there is a pocket. There is no interference with normal masticatory function, the tooth is firm in its socket, and has not drifted).	Horizontal bone loss involving the entire alveolar crest, up to half of the length of the tooth root (distance from apex to cemento-enamel junction)	6
Advanced destruction with loss of masticatory function (tooth may be loose, tooth may have drifted, tooth may sound dull on percussion with a metallic instrument, the tooth may be depressible in its socket)	Advanced bone loss, involving more than half of the length of the tooth root, or a definite intrabony pocket with definite widening of the periodontal membranes. There may be root resorption, or rarefaction at the apex	8

Scoring values (0, 1, 2, 6, and 8) relate to the stages of the disease scored in an epidemiological survey to the clinical condition observed. The jump from 2 to 6 in the scale recognizes the change in disease condition from a severe gingivitis to an overt destructive periodontal disease with obvious loss of attachment. PI can be considered a true interval scale.

Scores for each tooth are added, and the total is divided by the number of teeth examined. Scores can be interpreted as follows:

0.0–0.2 = Clinically normal supportive tissues.

0.3–0.9 = Simple gingivitis.

0.7–1.9 = Beginning destructive periodontal disease.

1.6–5.0 = Established destructive periodontal disease.

3.8–8.0 = Terminal periodontal disease.

Individual score = Average (scores for all of the teeth in the mouth)

Population score = Average (individual scores in population examined).

**Periodontal Disease Index (PDI).** The periodontal disease index was introduced by Sigurd P Ramfjord in 1959. It was a modification of Russell index, particularly designed for assessing the extent of pocket deepening below the cemento-enamel junction. The PDI comprises of three components

namely: Plaque component, Calculus component, Gingival & periodontal component. It combines the evaluation of gingival status with the probed attachment level (crevice depth measured from the cemento-enamel junction).

### **Selection of Teeth and Surfaces**

Teeth examined: (FDI system tooth numbers are in the parenthesis)

- Maxillary right first molar - (16)
- Maxillary left central incisor - (21)
- Maxillary left first bicuspid - (24)
- Mandibular left first molar - (36)
- Mandibular right central incisor - (41)
- Mandibular right first bicuspid - (44)

If any of the teeth are missing or unerupted, then only the teeth present are examined (only fully erupted teeth are used). Substitution is not made for missing teeth.

### *Scoring Method*

*For Gingival Status:* The gingiva around the teeth to be scored is first dried superficially by gently touching with absorbing cotton. Changes in color are evaluated by observing the color of the gingiva around the tooth to be scored and comparing the color corresponding to the buccal, lingual and interproximal surfaces with each other. Change in form is initially a blunting or rounding of the margin of the gingiva and thickening of papilla. Change in consistency is detected by applying gentle pressure with the side of periodontal probe against the gingiva to determine if there is soft or spongy consistency.

*For Crevicular measurements:* To measure crevice depth related to cemento-enamel junction, a University of Michigan #O Probe is used. The end of the probe should be placed against the enamel surface coronally to the margin of the gingiva so that the angle formed by the working end of the probe and long axis of the crown of the tooth is approximately 45°. Minimal force should be used to pass the probe in apical direction maintaining contact with the tooth. The probe should always be pointed towards the apex of the tooth or the central axis of multirooted teeth. After the distance from the free gingival margin to the CEJ has been measured, an attempt should be made to move the probe along the cemental surface. This can be achieved only if there has been loss of periodontal attachment. The University of Michigan number O probe is graduated at 3, 6 and 8 mm, making it necessary to estimate intervening measurements. The following criteria are used for crevicular measurements:

1. If the gingival margin is on enamel, measure from gum margin to CEJ and record the measurement. Then record the distance from the gingival margin to the bottom of the pocket. The distance from the CEJ to the bottom

of the pocket can then be found by subtracting the first from the second measurement.

2. If the gingival margin is on cementum, record the distance from the CEJ to the gingival margin as a minus value (a) then record the distance from the CEJ to the bottom of the gingival crevice as a positive value (b) Both loss of attachment and actual crevice depth can easily be assessed from these scores.

Ramfjord's method for measuring this distance is often referred to as the "Indirect method for measuring periodontal attachment loss".

Table 21. *Scoring Criteria: Periodontal Disease Index*

Gingivitis	No signs of inflammation	0
	Mild to moderate inflammatory gingival changes, not extending around the tooth	1
	Mild to moderately severe gingivitis extending all around the tooth	2
	Severe gingivitis characterized by marked redness, swelling, tendency to bleed and ulceration	3
Gingival crevice depth	Gingival crevice in any of the two measured areas (mesial, buccal), extend apically to the cementoenamel junction but not more than 3 mm	4
	Gingival crevice in any of the two measured areas extending apically to the cementoenamel junction from 3 to 6 mm inclusive.	5
	Gingival crevice in any of the two measured areas extending more than 6 mm apical to the cementoenamel junction	6

**Scoring PDI.**For individuals: Add the scores for individual teeth and divide by the number of teeth examined. The PDI ranges from 0 to 6.

*For group:* Total the individual PDI scores and divide by the number of individuals examined. The average ranges from 0 to 6. Plaque and Calculus Component of the Periodontal Disease Index (PDI). Although not part of PDI, a Plaque Index and Calculus Index are included when making a survey hence described.

**Dental Plaque.** For each of 6 teeth mentioned above 4 surfaces (facial, lingual, mesial and distal) are scored from 0 to 3.

#### **Procedure**

- Apply disclosing agent

- Patient is asked to expectorate and rinse with water
- Specific surfaces with disclosed plaque are observed.

Table 22. **Scoring Criteria for Plaque**

Criteria	Score
None	0
Present on some but not on all interproximal, buccal and lingual surfaces	1
Plaque present on all of the interproximal, buccal and lingual surfaces, but covering less than half of these surfaces	2
Plaque extends once all interproximal, buccal and lingual surface and covering more than one half of these surfaces	3

$$\text{Plaque score of an individual} = \frac{\text{Total score}}{\text{Number of teeth examined}}$$

**Calculus.** To measure the presence and extent of calculus a subgingival explorer or a periodontal probe is used.

**Procedure.** For each of the 6 teeth, the presence and extent of calculus on facial and lingual surfaces is scored from 0 to 3.

Table 23. **Scoring Criteria for Calculus**

Criteria	Score
None	0
Supragingival calculus, extending only slightly below the free gingival margin (not more than 1 mm)	1
Moderate amount of supra- and subgingival calculus or subgingival calculus alone	2
An abundance of supra- and subgingival calculus	3

**Scoring.For individual teeth:** Add scores for each surface and divide by the number of surfaces (4).

**For an individual:** Add the scores for an individual tooth and divide by the number of teeth.



## Question 5. X-ray diagnostics of periodontal diseases.

Table 24. X-ray diagnostics of periodontal diseases.

<b>Gingivitis</b>	X-ray changes in the marginal parts of the interalveolar septa are absent in most cases. There is a period of time when morphological bone changes are already present, but not yet detected on radiographs.
<b>Acute gingivitis</b>	Expansion of the marginal parts of the periodontal gap and focal osteoporosis of the cortical plates at the apexes of the interalveolar crests appear in the case of necrotizing ulcerative gingivitis forms in children and adolescents after 2-3 weeks.
<b>Chronic gingivitis</b>	As a rule, there are no radiological changes in the periodontium. Deposits of calculus are clearly visible. Sometimes, there is resorption of the compact plastic. The contours are saved. There is some widening of the periodontal gap in the area of the tooth neck.
<b>Periodontitis</b>	There is destruction of the bone tissue of the alveolar process (change in the contours, height and structure of the walls of the alveoli).
<b>Local periodontitis</b>	is characterized by a different degree of destruction of the interdental septum, expansion of the marginal parts of the periodontal gap. <i>On the radiograph:</i> "overhanging" fillings, improperly made artificial crowns, foreign bodies, large marginal carious cavities, subgingival dental deposits.
<b>Generalized periodontitis</b>	<p>Radiographically, three types of changes in the jaw bone tissue are distinguished:</p> <p><b>1. Destruction.</b> There is resorption of the bone tissue of the alveolar part of the jaw without spreading it to other parts of the jaw and without changing other bones of the skeleton, as a result of the inflammatory process.</p> <ul style="list-style-type: none"> <li>✓ Initial destruction. There is no compact plate of the apex of the interdental septa, osteoporosis without pronounced loss;</li> <li>✓ I degree. There is destruction of interdental septa up to 1/3 of the length of the tooth root;</li> <li>✓ II degree. There is destruction of interdental septa up to 1/2 of the length of the tooth root;</li> <li>✓ III degree. Destruction covers more than 2/3 of the interdental septum.</li> </ul> <p>This type of change is characteristic of chronic simple</p>

	<p>periodontitis.</p> <p><b>2. <i>Dystrophic changes</i></b> are expressed in sclerotic remodeling of bone tissue, combined with osteoporosis of the alveolar part and body of the jaw. Changes in other bones of the skeleton are also possible. There is a horizontal type of decrease in the height of the interdental septa. The second type is observed in the case of periodontal atrophy.</p> <p><b>3. <i>A combination of the two previous types</i></b>. It is characteristic of rapidly progressive periodontitis and chronic complex periodontitis.</p>
<b>Chronic simple periodontitis of mild severity</b>	<ul style="list-style-type: none"> <li>✓ osteoporosis of the interdental septa;</li> <li>✓ destruction of the cortical plate;</li> <li>✓ minimal bone resorption, not exceeding 20% of the total root length;</li> <li>✓ there are no changes in the furcation area.</li> </ul>
<b>Chronic simple periodontitis of moderate severity</b>	<ul style="list-style-type: none"> <li>✓ bone resorption up to 40% of the total root length;</li> <li>✓ changes in the furcation area (grade 2) a small or medium area of enlightenment between the roots;</li> <li>✓ the vertical type of bone tissue destruction prevails.</li> </ul>
<b>Chronic simple periodontitis, severe severity</b>	<ul style="list-style-type: none"> <li>✓ bone resorption more than 40% of the root length;</li> <li>✓ pronounced changes in the area of root furcation (extensive area of enlightenment in the interroot area)</li> <li>✓ bone resorption of a mixed type with a predominance of the vertical type of destruction of the alveolar ridge.</li> </ul>
<b>Chronic complex periodontitis</b>	<ul style="list-style-type: none"> <li>✓ uneven decrease in the height of the interdental septa, up to their disappearance;</li> <li>✓ a combination of horizontal and vertical types of bone resorption throughout the dentition;</li> <li>✓ alternation of foci of osteoporosis and osteosclerosis;</li> <li>✓ uneven expansion of the periodontal gap with a thickening of the compact plate in the apical region, in the furcation area;</li> <li>✓ resorption of a compact plate or its thickening;</li> <li>✓ hypercementosis;</li> <li>✓ root resorption;</li> <li>✓ cup-shaped bone atrophy</li> <li>✓ the presence of denticles in the tooth cavity;</li> <li>✓ migration of teeth (diastema, trema).</li> </ul>
<b>Juvenile periodontitis</b>	<ul style="list-style-type: none"> <li>✓ vertical resorption of the alveolar bone in the first molars and incisors;</li> </ul>

	<ul style="list-style-type: none"> <li>✓ arcuate loss of alveolar bone from the distal surface of the 2nd premolar and the medial surface of the 2nd molar (mirroring effect);</li> <li>✓ foci of resorption in the area of the remaining teeth join to symmetrical lesions in the case of generalized juvenile periodontitis;</li> <li>✓ progressive destruction of bone tissue is combined with the migration of teeth in the frontal area.</li> </ul>
<b>Periodontal atrophy</b>	There is functional restructuring of the marginal sections of the alveolar processes with a change in the shape and size of the interalveolar ridges while maintaining the anatomical integrity of these zones. A decrease in the height of the interalveolar septa from 1/3 to 1/2 of the length of the tooth root is observed in the case of dystrophic changes of mild and moderate severity in the horizontal type. Severe severity - more than 1/2 of the tooth root length.
<b>Idiopathic periodontal disease</b>	There is a mixed resorption of the bone tissue of the alveolar process with a predominance of the vertical type of resorption. There are many bone pockets.
<b>Papillon-Lefèvre syndrome</b>	<ul style="list-style-type: none"> <li>✓ lesion of the alveolar ridge with horizontal atrophy, resorption of the cortical plate,</li> <li>✓ bone pocket formation</li> </ul>
<b>Eosinophilic granuloma, oral manifestations</b>	There is bone resorption going from the alveolar edge to the jaw body. The focus of destruction has clear (sometimes unclear), not always even outlines. The vertical type of bone tissue destruction prevails.
<b>Hand-Schuller-Christian disease, oral manifestations</b>	<ul style="list-style-type: none"> <li>✓ resorption of the alveolar process throughout its entire length, oval or round,</li> <li>✓ cystic cavities with smooth edges,</li> <li>✓ the formation of bone pockets,</li> <li>✓ thinning of the cortical plate and trabeculae of the interalveolar septa.</li> </ul>
<b>Itsenko-Cushing's Disease</b>	<ul style="list-style-type: none"> <li>✓ foci of spongy osteoporosis</li> <li>✓ destruction of the alveolar bone</li> </ul> <p>The process can be localized in the base or body of the lower jaw and the alveolar part of the lower jaw.</p>
<b>Periodontal syndrome during diabetes mellitus</b>	is characterized by the presence of a funnel- and crater-like type of the bone tissue destruction of the alveolar process, which does not extend to the jaw body.

## **Radiographic Assessment of Periodontal Conditions**

Radiographs are especially helpful in the evaluation of the following features:

1. Amount of bone present
2. Condition of the alveolar crests
3. Bone loss in the furcation areas
4. Width of the periodontal ligament space
5. Local irritating factors that increase the risk of periodontal disease
  - Calculus
  - Poorly contoured or overextended restorations
6. Root length and morphology and the crown-to-root ratio
7. Open interproximal contacts, which may be sites for food impaction
8. Anatomic considerations
  - Position of the maxillary sinus in relation to a periodontal deformity
  - Missing, supernumerary, impacted, and tipped teeth
9. Pathologic considerations
  - Caries
  - Periapical lesions
  - Root resorption

## **Tests to the topic**

**1. Diagnosis of destructive processes in periodontal tissues should include:**

- a. An assessment of the general condition of the patient.
- b. Condition of the oral cavity.
- c. The detailed study of periodontal disease.
- d. All of the above.

**2. Indicate the criteria for implicating oral microorganisms as periodontal pathogens**

- a. The microorganism must be associated in high numbers in active periodontitis lesions and either absent (not cultivable) or in low numbers in gingivitis or healthy sites. The numbers of the microorganism should have increased to a threshold level before the onset of disease.
- b. The elimination of the microorganism, or its numerical reduction below threshold levels, should parallel remission of active disease.
- c. There should be a specific host immune response against the organism (i.e., elevated serum, salivary, and crevicular fluid antibody titers).
- d. The microorganism should evoke virulence factors that contribute to its pathogenicity or explain disease pathobiology.

- e. The microorganism should produce periodontitis in animal model systems.
- f. All of the above

**3. List the local anatomic risk factors**

- a. Furcation anatomy.
- b. Intermediate bifurcation ridges.
- c. Cervical enamel projections (CEP).
- d. Palato-gingival grooves (PGG).
- e. Open contacts and food impaction.
- f. Other anatomic risk factors of potential etiologic importance.
- g. All of the above

**4. List the iatrogenic risk factors**

- a. Overhanging dental restorations.
- b. Violation of the "biologic width".
- c. Open contacts and food impaction related to inadequate restorative dentistry.
- d. Occlusal traumatism.
- e. Additional local iatrogenic risk factors for periodontal diseases.
- f. All of the above.

**5. The goals of the patient examination of with periodontal pathology are**

- a. Assessment of the general condition of the body.
- b. The clinical characteristics of periodontal disease.
- c. The identification of common and local etiological and pathogenetic factors, the definition of the form.
- d. Stage and nature of the course disease.
- e. All of the above.

**6. Examination of the dentition consists of**

- a. The number of removed teeth.
- b. Hypersensitivity of dentin.
- c. The condition of approximal contact of the teeth.
- d. Mobility.
- e. Percussion of teeth.
- f. The presence of pathological migration.
- g. Occlusal contacts.
- h. All of the above.

**7. Periodontal indices include**

- a. CPI, CPITN.

- b. DFMT.
- c. PI, PDI.
- d. GI.
- e. OHIS, PHP.

**8. What will the radiograph show in case of chronic simple periodontitis of moderate severity?**

- a. Bone resorption up to 40% of the total root length.
- b. Changes in the furcation area (grade 2) a small or medium area of enlightenment between the roots.
- c. The vertical type of bone tissue destruction prevails.
- d. All of the above.

**9. What will the radiograph show in case of juvenile periodontitis?**

- a. Vertical resorption of the alveolar bone in the first molars and incisors.
- b. Arcuate loss of alveolar bone from the distal surface of the 2nd premolar and the medial surface of the 2nd molar (mirroring effect).
- c. Foci of resorption in the area of the remaining teeth join to symmetrical lesions in the case of generalized juvenile periodontitis.
- d. Progressive destruction of bone tissue is combined with the migration of teeth in the frontal area.
- e. All of the above

**10. Pocket involving two or more tooth surfaces is called**

- a. Simple pocket.
- b. False pocket.
- c. Compound pocket.

## **LESSON 8. DENTAL CARIES.DEVELOPMENT MECHANISM. EPIDEMIOLOGY. CLASSIFICATIONS.**

The questions to be studied for the learning of the topic:

1. Dental caries. Definition. Theories of caries appearance.
2. Etiology of dental caries. Microbiology of caries. The concept of dental plaque, role of biofilm.
3. Pathogenesis of dental caries.
4. Histopathology of dental caries.
5. Epidemiology of dental caries.
6. Caries classifications. Black's classification. International diseases classification. Anatomical classification.

### **Question 1. Dental caries. Definition. Theories of caries appearance.**

Dental caries is an irreversible microbial disease of the calcified tissues of the teeth, characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth, which often leads to cavitations. The word caries is derived from the Latin word meaning 'rot' or 'decay'. It is a complex and dynamic process where a multitude of factors initiate and influence the progression of disease.

Caries is still a major oral health problem in most industrialized countries, affecting 40-90% of schoolchildren and about 90% of adults.

Caries is the most prevalent oral disease in several Asian, European and Latin American countries.

The etiology of dental caries is generally agreed to be a complex problem complicated by many indirect factors that obscure the direct cause or causes. There is no universally accepted opinion for the etiology of dental caries. Numerous references on dental caries, including early theories attempting to explain its etiology, have been found in recorded history of ancient people. However, many theories have evolved through years of investigation and observation; the acidogenic theory of Miller (Miller's chemico-parasitic theory), the proteolytic theory and the proteolysis chelation theory, are among those which have stood the test of time.

#### **The early theories**

***The Legend of Worms.*** The earliest reference to tooth decay is probably from the ancient Sumerian text known as the «Legend of Worms» from about 5,000 BC. The idea that caries is caused by worms was possibly prevalent for a long time as evident from the writings of Homer who made reference to worms as the cause of toothache.

***Endogenous theories.*** Keeping with the humoral theory of Greek physicians, dental caries was thought to be produced by internal action of acids and corroding humors. Along with this, the early Greek physicians such

as Hippocrates, Celsus, and Galen, proposed the vital theory of tooth decay, which postulated that tooth decay originated, like a bone gangrene, from within the tooth itself.

***Chemical theory.*** Parmlly in 1820s observed that dental decay affected externally, not internally, as had been thought previously. It was proposed that an unidentified 'chymal agent' was responsible for caries. This was further supported by Robertson in 1835 who proposed that dental decay was caused by acid formed by fermentation of food particles around the teeth.

***Parasitic theory.*** The first to relate microorganisms to caries on a causative basis as early as 1843 was Erdl who described filamentous organisms in the membrane removed from teeth. Shortly thereafter, Ficinus in 1847, a German physician in Dresden, attributed dental caries to 'denticolae' the generic term he proposed for decay related microorganisms. Leber and Rottenstein, two German physicians, disseminated the idea that dental caries commenced as a chemical process but that living microorganisms continued the disintegration in both enamel and dentin. In addition to these observations, Clark (1871, 1879), Tomes (1873) and Magitot (1878) concurred that bacteria were essential to caries, although they suggested an exogenous source of the acids. In 1880, Underwood and Miller presented a septic theory with the hypothesis that acid capable of causing decalcification was produced by bacteria feeding on the organic fibrils of dentin. They reported sections of decayed dentin having micrococci as well as oval and rod shaped forms.

***Miller's chemico-parasitic theory or the acidogenic theory.*** The chemico-parasitic theory is a blend of the above mentioned two theories. Willoughby D Miller, an American who was working at the University of Berlin, is probably the best known of the early investigators on dental caries. He published extensively on the results of his studies, beginning in 1882, which culminated in the hypothesis, "Dental decay is a chemico-parasitic process consisting of two stages, the decalcification of enamel, which results in its total destruction and the decalcification of dentin as a preliminary stage, followed by dissolution of the softened residue. In case of enamel; however, the second stage is practically wanting, the decalcification of enamel signifying its total destruction". The acid, which affects this primary decalcification, is derived from the fermentation of starches and sugar lodged in the retaining centers of the teeth. Miller found that bread, meat and sugar incubated in vitro with saliva at body temperature, produced enough acid within 48 hours to decalcify sound dentin. Subsequently, he isolated numerous microorganisms from the oral cavity, many of which were acidogenic and some were proteolytic. Since a number of these bacterial forms were capable of forming lactic acid, Miller believed that caries was not caused by any single organism, but rather by a variety of microorganisms. He



assigned an essential role to three factors in the caries process: the oral microorganisms in acid production and proteolysis; the carbohydrate substrate; and the acid which causes dissolution of tooth minerals. Miller's chemico-parasitic theory is the backbone of current knowledge and understanding of the etiology of dental caries. However, Miller's chemico-parasitic theory could not explain the predilection of specific sites on a tooth to dental caries and the initiation of smooth surfaces. Also, why some populations are caries-free and the phenomenon of arrested caries.

So, Miller's chemico-parasitic theory or acidogenic theory:

1. Caries is caused by acids produced by microorganisms of the mouth.
2. Dental decay is a chemico-parasitic process consisting of two stages:
  - Decalcification of enamel and dentin (preliminary stage)
  - Dissolution of the softened residue (subsequent stage). Acids resulting in primary decalcification are produced by the fermentation of starches and sugar from the retaining centers of teeth.

The concept of dental plaque adhering to teeth and serving to localize bacterial enzymatic activity was proposed later in 1897 by Williams. This theory has been accepted by majority of investigators in a form essentially unchanged since its inception. The bulk of scientific evidence does implicate carbohydrates, oral microorganisms and acids, and for this reason, these deserve further consideration.

### **Question 2. Etiology of dental caries. Microbiology of caries. The concept of dental plaque, role of biofilm.**

In 1920s Clarke described a spherical bacterium that formed chains of cells, isolated from dental caries lesions. He named this organism *Streptococcus mutans* (different morphological forms which he believed were mutants). *S. mutans* produced lactic acid as a main by-product from glucose fermentation (homolactic). But there was a strong cohort of *Lactobacillus* microbiologists at the time, and Clarke's attempts to have *Streptococcus* named as a new genus were foiled.

By the late 1950s, Keyes and Fitzgerald began working on the nature of this transmissible factor. A *Streptococcus* was purified from carious lesions of hamsters (also from rats) that was strongly acidogenic (producing acid) and non-proteolytic.

It was not until 1968 that it was accepted that the *Streptococcus* isolated from hamsters was the same *S. mutans* as that described by Clarke in 1924.

***Streptococcus mutans*.** Gram-positive cocci in chains. More accurately, a collection of closely related species known as mutans streptococci and comprising seven species and eight serotypes, a–h. *S. mutans* serotypes c, e and f and *S. sobrinus* serotypes d and g are most closely associated with

human disease. *S. cricetus*, *S. ferus*, *S. rattus*, *S. macacae* and *S. downei* are more usually found in animals. Mutans streptococci possess adhesins for salivary receptors allowing attachment to saliva-coated smooth surfaces. In addition, these organisms produce extracellular polysaccharides from sucrose that facilitate retention on surfaces. Mutans streptococci are associated with all forms of caries.

**Virulence factors of *S. mutans*.** *S. mutans* possess several attributes that contribute to its success as a cariogenic organism: (1) ability to adhere to the tooth surface and develop plaque communities; (2) production of glucans and other polysaccharides from excess carbohydrate (often sucrose) in the diet, leading to plaque accumulation; (3) production of acids (principally lactic acid), that generate a low pH environment and enrich for aciduric organisms.

*S. mutans* is adapted to the biofilm lifestyle and there is coordinated production of bacteriocins along with an increase in competence in high density situations. *S. mutans* may thus acquire DNA from other organisms in close proximity either for nutrition or increasing genetic diversity or both.

*S. mutans* can metabolize a variety of sugars, resulting in the production of a number of weak acids, including lactic, formic and acetic acids. Lactic acid is the strongest of these acids, with an ionization constant (pKa) of 3.5. When the plaque pH drops below about 5.5 the balance between enamel demineralization and remineralization shifts toward solubility and the caries process is initiated.

*S. mutans* produces polymers of glucan and fructan from dietary sucrose through glucosyl- and fructosyl-transferases.

*S. mutans* participates in Initial attachment to tooth surfaces producing a major surface protein. A major surface protein is the AgI/II family protein called SpaP (or P1). This protein contributes to the fibrillar layer that is on the outside of *S. mutans* cells, made up of proteins, polysaccharides and teichoic acids.

*S. mutans* is highly aciduric, and resistance to the adverse effects of low pH.

Organisms such as Lactobacilli that produce and tolerate large amounts of lactic acid are not thought to be the initiators of smooth surface caries as they lack specialized colonization mechanisms. Actinomyces and non-mutans streptococci produce less acid and so may be more important in root caries as cementum (and dentin) is less mineralized and thus more easily dissolved, as compared to enamel.

**Lactobacilli.** Gram-positive rods lactobacilli are efficient producers of lactic acid and are tolerant to low pH values (two important caries associated traits). However, lactobacilli are poor colonizers of smooth surfaces and probably do not initiate caries at these sites. Most likely lactobacilli are

secondary colonizers of established caries lesions, where their aciduric properties allow them to out compete other organisms.

Acid production will then exacerbate the lesion and facilitate extension into the dentin. If lactobacilli become embedded in pits and fissures they may be able to initiate caries at these sites. Different species and strains of lactobacilli exhibit differing cariogenic potential.

***Actinomyces species.*** Gram-positive rods *Actinomyces*, especially *A. naeslundii*, are frequently isolated from root caries lesions and can cause root caries in experimental animals. However, the organisms are also commonly found on healthy root surfaces so the role of *actinomyces* in the disease process has been unclear. More recent molecular detection techniques are re-establishing the importance of *Actinomyces species* in both root and coronal caries.

### **Question 3. Pathogenesis of dental caries.**

#### **Demineralization of enamel. The role of critical pH.**

Critical pH is the term given to the highest pH at which there is a net loss of minerals from tooth enamel. This is the pH at which saliva and plaque fluid cease to be saturated with calcium and phosphate, thereby permitting hydroxyapatite to dissolve. Critical pH is generally accepted to be 5.5, but it can be a little higher or lower depending on individual factors. During the demineralization process, acid diffuses between the rods and reaches deeper areas of the enamel and into dentin, where carbonated hydroxyapatite crystals are more susceptible to dissolution. The calcium and phosphate ions that are lost from the tooth diffuse out into dental plaque fluid and saliva. If the acid attack is chronic and prolonged, progressively greater amounts of calcium and phosphate minerals diffuse out of the tooth, causing the crystalline structure of the tooth to shrink in size, while pores enlarge. Eventually, a carious lesion develops; its rate of development is a function of the degree of undersaturation of fluid in its environment and rates of diffusion of ions into and out of enamel.

#### **Host and environmental factors in caries.**

Dental caries is multifactorial disease in which there is an interaction among three principle factors

1. Susceptible host tissue
2. Cariogenic microflora
3. Suitable local substrate - fermentable carbohydrates

The initiation and progression of caries requires that host, diet and bacterial factors are all conducive to disease.

## *Host factors*

### 1. Teeth

#### **1) Composition and structure of enamel**

- Composition of surface enamel due to dense mineralization and high F-content, surface enamel more resistant to caries than subsurface layers of enamel
- Type of crystalline lattice of enamel.

Teeth become less susceptible to caries over time. Such post-eruptive resistance is due in part to an increase in the concentration of fluoride in the surface layer of enamel. Fluoride ions substitute for hydroxyl ions in hydroxyapatite, forming fluorapatite which is less soluble in acid than hydroxyapatite.

**2) Tooth morphology** contributes to caries susceptibility on the basis of ease of bacteria colonization. Deep, narrow occlusal, buccal and lingual pits promotes to trap food, bacteria or debris.

**3) Tooth position** rotated, misaligned teeth, deformed dentitions are difficult for cleanse and tends to accumulation of food and debris

### 2. Saliva

**1) Flow rate.** The flow of saliva physically washes away weakly attached bacteria and acids, and delivers salivary buffers. Xerostomia (low salivary flow < 0.1 ml/min) leads to rampant caries. Impaired salivary flow is deleterious to oral health. For example, an inadequate salivary flow rate increases the incidence of dental caries, for at least three reasons. First, there is greater bacterial retention in the mouth and more dental plaque forms; second, the acids produced by bacteria such as mutans group streptococci are inefficiently neutralized; and third, the enamel surface does not efficiently re-mineralize.

Around 0.5 to 1.5 liters of saliva are secreted into the mouth each day.

Saliva is responsible for flushing the epithelial surfaces and for lubrication and protection of tissues and an adequate flow of saliva is essential for the maintenance of both hard and soft tissue integrity. Saliva is hypotonic, with an average pH of around 6.7. Saliva contains both organic compounds (2–3 g/l protein, notably the enzyme amylase) and inorganic compounds including the electrolytes bicarbonate, chloride, potassium and sodium.

Salivary flow rate and composition can be affected by a range of infectious diseases, clinical conditions, e.g. wearing of dentures, clinical treatments, e.g. radiation therapies for oral cancer, or pharmaceutical drugs.

Saliva also contains a number of anti-microbial compounds that can restrict plaque accumulation and kill bacteria and other microorganisms.

**2) Buffering capacity.** Saliva has two major buffering systems: bicarbonate-carbonic acid  $\text{HCO}_3^-/\text{H}_2\text{CO}_3$  (is the most important) and phosphate.

Buffering by saliva helps prevent bacterial acids, from reducing the pH to levels that dissolve apatite.

**3) Protective role of saliva.** There are between 1 million and 100 million bacteria present in 1 ml of saliva, depending upon oral hygiene, frequency of food consumption, and salivary flow rate. Saliva in the fluid phase acts principally to flush out bacteria from the mouth. Saliva contains agglutinins that aggregate bacteria, thus preventing adherence to surfaces, and the bacterial clumps are removed by swallowing or expectoration.

#### *Anti-microbial components in saliva*

**(1) Lysozyme.** Lysozyme is a basic protein found in most secretions, including saliva, where it is present in high concentrations. Salivary lysozyme originates from both the salivary gland secretions and from gingival crevicular fluid (GCF). Lysozyme digests the cell walls of Gram-positive bacteria by breaking the bond between N-acetylmuramic acid and N-acetylglucosamine in peptidoglycan. Lysozyme can also activate autolysins in bacterial cell walls. Not surprisingly, many successful oral colonizers are relatively resistant to killing by lysozyme. Lysozyme can also bind and aggregate bacteria and facilitate clearance by swallowing or expectoration. In addition, lysozyme contains small amphipathic sequences in the C-terminal region that are capable of killing bacteria.

**(2) Salivary peroxidase.** Peroxidase in saliva is derived from the salivary glands and PMNs, and catalyzes the oxidation of thiocyanate (SCN) to hypothiocyanite (OSCN) by hydrogen peroxide, which is produced by the aerobic metabolism of oral bacteria. At acid pH, OSCN becomes protonated (and uncharged) and readily passes through bacterial membranes. Hypothiocyanite oxidizes SH groups in bacterial enzymes and inhibits bacterial metabolism. Reduction of hydrogen peroxide to water by peroxidase also prevents oxidative damage to the host soft tissues.

**(3) Lactoferrin.** Lactoferrin is an iron binding glycoprotein produced from glandular acinar cells, epithelial cells and phagocytic cells. Lactoferrin inhibits bacterial growth by binding and sequestering Fe<sup>2+</sup> ions, and in the apo (iron free) state can be toxic to bacteria and interfere with bacterial adhesion. A 25-residue N-terminal proteolytically derived peptide fragment termed lactoferricin also kills bacteria through depolarization of cytoplasmic membranes.

**(4) Histatins.** Histatins are cationic histidine rich proteins that kill *Candida albicans* and some bacteria.

**(5) Cystatins.** Cystatins are cysteine rich peptides that inhibit bacterial cysteine proteases.

**(6) Chromogranin A.** Chromogranin A is produced by the submandibular and sub-lingual glands, and is processed to release an N-

terminal peptide, vasostatin-1, which is antibacterial and antifungal protein from epithelial cells.

(7) **Immunoglobulins** (IgA from saliva, IgG and IgM from serum via GCF).

**4)Supersaturation** of saliva with calcium, phosphate and fluoride allows remineralization of enamel. At physiological pH saliva is supersaturated with respect to calcium and phosphate, that helps prevent loss of calcium and phosphate from enamel mineral.

### **3. Systemic factors**

- Heredity
- Pregnancy and lactation.

### **4. Dietary factors**

In order to produce acid, cariogenic bacteria require a fermentable carbohydrate substrate, in particular sucrose. Studies have shown that in addition to total consumption, the frequency of intake and physical form of the sucrose containing food are important. The potential cariogenicity of food can be assessed by measuring the pH changes in plaque over time following ingestion. In order to produce acid, cariogenic bacteria require a fermentable carbohydrate substrate, in particular sucrose.

Sucrose is the most cariogenic sugar because:

- it can be processed into glucan and fructan
- it is efficiently fermented into lactic acid
- sucrose and other sugars are transported into SM cells by the high affinity and high capacity phosphoenolpyruvate (PEP) sugar: phosphotransferase (PTS) uptake system
- has the additional cariogenic property of providing a substrate for bacterial glucosyltransferases and fructosyltransferases

***Role of carbohydrates.*** Reference has been made previously to the finding that members of isolated primitive societies who had a relatively low caries index manifested a remarkable increase in caries incidence after exposure to refined diets. The presence of readily fermentable carbohydrates has been thought to be responsible for their loss of caries resistance. The early studies of Miller showed that when teeth were incubated in mixtures of saliva and bread or sugar, decalcification occurred. There was no effect on the teeth when meat or fat was used in place of the carbohydrate. Both cane sugar and cooked starches produced acid, but little acid was formed when raw starches were substituted. Volker and Pinkerton reported the production of similar quantities of acid from mixtures of either sucrose or starch incubated with saliva with no difference in acid production between raw and refined sugar cane. The etiology of dental caries involves interplay between oral bacteria, local carbohydrates and the tooth surface that may be shown as follows: Bacteria + sugars+ teeth→organic acids→caries.

The cariogenic carbohydrates are dietary in origin, since uncontaminated human saliva contains only negligible amounts regardless of the blood sugar level. Salivary carbohydrates are bound to proteins and other compounds, and are not readily available for microbial degradation. The cariogenicity of a dietary carbohydrate varies with the frequency of ingestion, physical form, chemical composition, route of administration and presence of other food constituents. Sticky, solid carbohydrates, soft retentive foods those that are cleared slowly, monosaccharides and disaccharides are more caries-producing. Plaque organisms produce little acid from the sugar alcohols, sorbitol, and mannitol. Glucose or sucrose fed entirely by stomach tube or intravenously, does not contribute to decay as they are unavailable for microbial breakdown. Meals high in fat, protein or salt reduce the oral retentiveness of carbohydrates.

#### **Question 4. Histopathology of dental caries.**

##### Zones of enamel caries:

- The early (submicroscopic) lesion
- Phase of nonbacterial enamel crystal destruction
- Cavity formation
- Bacterial invasion of enamel.

**Early submicroscopic lesion.** In early lesion, earliest visibility changes appear as white opaque spot on the surface of tooth and adjacent to contact point other than this chalking white appearance the enamel is hard and smooth on appearance. This caries is known as incipient caries.

1st observable changes occur in this **translucent zone (1)**. It appears as by formation of submicroscopic spaces or by pores locate at the prism boundaries and other junctional sites such as on the stiae of Retzius. Here 1% demineralization occurs.

**2. Dark zone.** It lies adjacent to translucent zone .It is defined as positive zone because it is always present. This zone is formed by demineralization and here 2-4% demineralization occurs.

**3. Body of the lesion.** It lies between surface zone and body of the lesion. Here greatest amount of demineralization occur. Here 5% demineralization occurs near the periphery and 25 % demineralization occur on the center of the lesion.

**4. Surface zone.** When it is examined by polarized microscope, micro radiography shows relatively unaffected. The greater resistance of this layer may be due to greater amount of remineralization or may be due to greater concentration of surface enamel.

Once bacteria have penetrated the enamel they reach the amelodentinal junction and spread laterally to undermine the enamel. This has three major effects. First, the enamel loses the support of the dentine and is therefore

greatly weakened. Second, it is attacked from beneath. Third, spread of bacteria along the amelodentinal junction allows them to attack the dentine over a wide area. Thus the primary lesion provides the bridgehead for the attack on enamel, but undermining of the enamel determines the area of a cavity. Clinically this is frequently evident when there is no more than a pinhole lesion in an occlusal pit, but cutting away the surrounding enamel shows it to be widely undermined. As undermining of the enamel continues, it starts to collapse under the stress of mastication and to fragment around the edge of the (clinically obvious) cavity. By this stage, bacteria damage to the dentine is extensive.

### **Question 5. Epidemiology of dental caries.**

***Caries in Prehistoric Man.*** Dental caries is probably a disease of modern civilization. Anthropologic studies of von Lenhossek revealed that the dolichocephalic skulls of men from preneolithic periods (12,000 BC) did not exhibit dental caries, but brachycephalic skulls of the neolithic period (12,000–3000 BC) contained carious teeth. Apparently the carious lesions were found at or just below the contact areas and an increased frequency of caries at the cemento-enamel junction was noted.

***Caries incidence in modern societies.*** By about the 17th century, there was a significant increase in the total caries experience and a smaller increase in the number of carious lesions involving the interproximal contact areas of teeth, characteristic of the pattern and occurrence of caries in modern population. Extensive studies on the incidence of dental caries from various geographic areas have illustrated the apparent influence of civilization on dental disease. Mellanby in 1934 reviewed the literature on caries in existing primitive races and noted that the incidence was invariably less than that in modern man suggesting isolated populations that have not acquired the dietary habits of modern, industrialized man retain a relative freedom from dental caries. Native population living in the North West territories of Canada, Alaska and Greenland who consumed native food, had a lower evidence of carious lesion (0.1%) compared to those living at trading posts (13%). A comparable effect of diet upon caries was demonstrated by Mellanby in studies on natives of Southern Rhodesia. The determinants of the carious process are essentially local and limited to the oral cavity. Although there may be a certain degree of racial resistance to dental caries, dietary factor appears to be more significant, especially since caries incidence is increased by contact with 'civilized' food.

***DMF AND def INDEX.*** The most commonly employed method to measure the extent of previous damage to permanent dentition is by a measure known as the DMF index. The designation DMF (T) is used to denote decayed, missing, and filled teeth; DMF(S) denotes decayed, missing



and filled surfaces in permanent teeth and therefore takes into account the number of surfaces attacked on each tooth. A similar index def (t) or def (s) index is used for primary dentition. The DMF/def index can be used to quantify both caries prevalence and caries incidence in a given population. It is an arithmetic index of the cumulative caries attack in a population. A commonly used modified form of this test is the caries increment, which refers to the number of new carious lesions occurring in a specified time interval, either for an individual or averaged over a population. The assessment of the caries increment involves at least two examinations—one at the beginning and one at the end of the period in question. In children, primary teeth may be lost due to natural exfoliation and, for the purpose of the def index, it is essential that the examiner designates as missing only those teeth that are lost due to caries.

The oral health of children 12 years old is the object of several epidemiological studies conducted around the world. According to the World Health Organization (WHO, 1997), the importance given to this age group is due to the fact that it is this age that children leave primary school. Thus, in many countries, is the last age at which data can be easily obtained through a reliable sample of the school system. Moreover, it is possible that at this age all the permanent teeth except third molars, have already erupted. Thus, the age of 12 was determined as the age of global monitoring of caries for international comparisons and monitoring of disease trends. Even considering the large number of scientific evidence from several epidemiological studies in schoolchildren worldwide, the majority are regional studies. In addition, the information is too outdated for some countries, which does not make easy international comparison. The index that measures the number of permanent teeth decayed; missing and filled teeth (DMFT) is the common outcome for such studies.

The WHO Oral Health Program (Petersen, 2003) presented in its report on the global oral health conditions, a four-level scale for the classification of the DMFT index at 12 years-old. They are: very low (less than 1.2), low (1.2 to 2.6), moderate (2.7 to 4.4) and high (over 4.4).

### **Factors Affecting Caries Prevalence**

**Race.** Some studies show remarkable differences in the caries experience between races. American blacks and whites, living in the same geographic areas under similar conditions, offer an excellent opportunity for comparison. Investigations indicate that the blacks have fewer carious lesions than the whites. Most studies concerning other races have been relatively unsatisfactory because of complicating factors such as differences in diet or exposure to fluoride, which tend to mask any differences due to racial background. Nevertheless, there is some evidence to indicate that blacks, Chinese, and East Indians have considerably less caries than American

Whites. The English have a higher caries incidence than Italians, Russians, and Chinese.

**Age.** Carious lesions that result in cavitation are irreversible and therefore, cumulative with age. There is a strong correlation between age and DMF indices. Several studies have shown that by the age of 6 years, about 20% of children have experienced dental caries in their dentition and a DMFT of 0.5 can be expected. By the age of 12 years, 90% of children would have experienced a DMFT of approximately 5.5. The decayed, missing and filled surface (DMFS) accelerates at a greater rate than the DMFT beyond the age of eight years. By the age 12, an average DMFS of 7.5 is seen in most populations. In general, other reports of caries prevalence among children in various parts of the world show rates that seem to be comparable to those cited here. Another common element is that children from families in lower socioeconomic groups consistently have greater caries prevalence than their peers from families at a higher socioeconomic level.

**Gender.** Studies indicate that the total caries experience in permanent teeth is greater in females than in males of the same age. This is attributable largely to the fact that the teeth of girls erupt at an earlier age. This time difference is particularly significant during the formative years because teeth have been shown to be maximally susceptible to dental caries immediately after eruption since, the chemical structure of teeth in the immediate post eruptive stage is suboptimal in terms of caries resistance. As teeth are exposed to saliva and constituents in the diet, the outer layers of the tooth take up additional minerals from the oral environment in a process known as posteruptive maturation. This maturation process confers a greater resistance to dental caries on the tooth.

**Familial.** Siblings of individuals with high caries susceptibility are also generally caries active, whereas siblings of caries immune individuals generally exhibit low caries rates. Children of parents with a low caries experience also tend to have low caries; the converse is true for children whose parents have a high caries rate. Studies of the dental caries experience in monozygotic and dizygotic twins indicate that concordance for carious sites in monozygotic twins is much higher than in dizygotic twin pairs.

## **Question 6. Caries classifications. Black's classification.**

### **Anatomical classification. IDC.**

The different features of dental caries are differentiated.

#### ***1. Anatomical caries classification is according to morphological site:***

- ✓ Pit and fissure caries
- ✓ Smooth surface caries.
- ✓ Cemental or root caries

Pit and fissure caries develops in:

- ✓ Occlusal surface of molar and premolar.
- ✓ Buccal surface of molars.
- ✓ Lingual surface of maxillary incisors.

Deep pits and fissures are naturally more prone to caries due to their poor self cleansing properties and tendency to entrap food debris and bacteria. Also, enamel at the base of such pits and fissures is thin. Early caries appears brown or black discoloration which is rough in probe. Staining is due to pigmentations from tobacco and bacteria. Enamel bordering the pit appears bluish-white as it is undermined. Upon reaching the DEJ, there is lateral spread of caries and penetration of dentinal tubules. All this occurs without apparent fracturing away of overlying enamel. Thus there may be a large carious lesion inside with only a pinpoint opening seen on the tooth surface. Sometimes caries begins as open cavity, this type is slower.

**Smooth surface caries** develops in:

1) *Proximal surface*. Here, the caries is preceded by formation of dental plaque, unlike the pit and fissure caries. Presence of plaque ensures retention of carbohydrate and bacteria on tooth surfaces, leading to subsequent acid production and demineralization of enamel.

Smooth surface caries usually begins just below the contact point and appears in initial stages as a faint white opacity of enamel without loss of continuity of enamel surface. The early white spot becomes roughened due to superficial decalcification of enamel.

As caries reaches the DEJ, there is rapid lateral spread.

2) *Gingival third of buccal and lingual surface (cervical caries)*. It always occurs as an open cavity unlike the smooth surface or pit and fissure caries. It occurs on all the teeth without predilection as it is directly related to lack of oral hygiene.

**Root caries.** Root caries is concerning for the elderly population who often have gingival recession exposing the root surfaces and they have also reduce salivary flow.

Carious lesions form more quickly on root surfaces than coronal caries because the cementum on the root surface is softer than enamel and dentin. Microorganisms invade cementum along Sharpey's fibers.

## **2. Classification according to rapidity of caries:**

Caries is divided into:

1) *Acute dental caries*. It runs rapidly so it leads to early pulp exposure. It occurs in young adults because the dentinal tubules are large and so, there is little time for secondary dentin formation. The entrance of carious cavity is small. The small point of opening doesn't allow the buffering ions of saliva to neutralize acids formed within the cavity.

**Rampant caries:** It is the sudden, rapid destruction of teeth affecting even relatively caries free surfaces like proximal and cervical surfaces of

mandibular teeth. 10 or more carious lesions over a one year period is characteristic of rampant caries.

***Nursing bottle caries.*** It is a rampant caries affecting deciduous teeth in babies due to prolonged use of milk after eruption of deciduous teeth.

***Radiation caries:*** It is a type of rampant caries occurring in patients receiving radiotherapy in head & neck region. Xerostomia is a major complication of radiotherapy of head and neck.

2) ***Chronic dental caries.*** This type of caries is slowly progressive. It occurs in adult. The entrance for caries is large. So, there is less food retention and greater saliva access. This allows time for sclerosis of dentin and deposition of secondary dentin.

3) ***Arrested caries.*** Caries which becomes static. This occurs in case of:

- Occlusal surface with large cavity, so there is lack of food retention which leads to hard polished surface (eburnation of dentin). Secondary dentin is formed.
- In proximal surface if adjacent tooth is extracted.
- Following topical application of fluoride.

### **3. Caries classification according to onset of occurrence:**

Caries is divided into:

a) ***Primary caries.*** Caries that occurs for the first time.

b) ***Secondary (recurrent) caries.*** Secondary, or recurrent, caries starts to form in the small spaces or gaps between the tooth and the margins of a restoration.

### **4. Caries classification Based on tissue involved:**

- ✓ Enamel caries
- ✓ Dentin caries
- ✓ Cemental caries

### **5. Classification of caries lesions. Black's classification:**

***Lesions of class I.*** Locations include:

- Occlusal surface of molars and premolars
- Lingual surface of anterior teeth
- Occlusal two thirds of buccal and lingual surfaces of molars and premolars, i.e. blind pits of teeth

***Lesions of class II.*** Lesions occur on the proximal surfaces of the posterior teeth - molars and premolars.

***Lesions of class III.*** Lesions occur on the proximal surfaces of anterior teeth – incisors and canines. Class III cavities do not involve an incisal angle.

***Lesions of class IV.*** Lesions occur on the proximal surfaces of anterior teeth when the incisal angle is involved and requires restoration.

***Lesions of class V.*** Lesions occur on smooth facial and lingual surfaces in gingival third of teeth. Lesions begin close to gingival may involve a cementum or dentinal surface as well as enamel.

*Lesions of class VI.* Involve the incisal edges of anterior teeth and the occlusal surfaces posterior teeth that have been worn away due to abrasion.

**International classification ICD-10**

K02 Dental caries

K02.0 Caries limited to enamel. White spot lesion [initial caries]

K02.1 Caries extending into dentine

K02.2 Caries of cementum

K02.3 Arrested caries

K02.4 Odontoclasia

K02.8 Other specified dental caries

K02.9 Dental caries, unspecified

**Tests to the topic**

**1. What acid is *Streptococcus mutans* capable of metabolizing, and in the process, further promoting demineralization?**

- a. Lactic acid.
- b. Acetic acid.
- c. Pyruvate acid.
- d. Formic acid.
- e. Hydrochloric acid.

**2. Which factors affect the rate at which acid is produced in plaque?**

- a. The microbial composition of the dental plaque.
- b. The density of plaque.
- c. The speed at which bacteria are able to metabolize the dietary carbohydrate.
- d. All of the above.

**3. At what pH does tooth enamel begin to demineralize?**

- a. 8.3.
- b. 7.5.
- c. 5.5.
- d. 3.2.

**4. What is the clinical appearance of the initial stage of a carious lesion?**

- a. A large cavitation that extends into the dentin.
- b. A chalky white and softened spot on the tooth surface.
- c. Evidence of tooth erosion caused by acid attack.
- d. Completely demineralized tissue.

**5. Which of the following is a remineralization-promoting characteristic of saliva?**

- a. Saliva promotes acidity that promotes hydroxyapatite crystal growth.
- b. Saliva does not promote remineralization.
- c. Saliva is supersaturated with calcium and phosphate ions.
- d. None of the above.

**6. How is the biological hydroxyapatite of tooth enamel different than pure hydroxyapatite?**

- a. It readily incorporates trace minerals, such as fluoride and carbonate into its crystal lattice.
- b. It is stronger.
- c. It has the following formula:  $\text{Ca}_{12}(\text{PO}_4)_8(\text{OH})_4$
- d. All of the above.

**7. Which of the following is true about fluorapatite?**

- a. It has crystal structure resulting from the replacement of hydroxyl ions ( $\text{OH}^-$ ) in the hydroxyapatite structure with fluoride ions.
- b. Crystal structure of fluorapatite is a more compact structure than hydroxyapatite.
- c. It is stronger and more acid resistant than hydroxyapatite.
- d. All of the above.

**8. A cavity which located on the proximal surface of molars and premolars is classified as:**

- a. Black's Class I.
- b. Black's Class II.
- c. Black's Class III.
- d. Black's Class IV.
- e. Black's Class V.

**9. Which of the following is true about acute caries?**

- a. It runs rapidly so it leads to early pulp exposure
- b. The entrance of carious cavity is small
- c. Lesions are dark or brown
- d. Lesions are soft and are light colored

**10. What index can be used to quantify both caries prevalence and caries incidence in a given population?**

- a. DMFT.
- b. GI.
- c. PI.
- d. PMA.

## **LESSON 9. ENAMEL CARIES. CLINICAL APPEARANCE. DIAGNOSTICS. TREATMENT**

The questions to be studied for the learning of the topic:

1. Composition of enamel caries.
2. Histopathology of enamel caries.
3. Clinical appearance of enamel caries.
4. Main and additional methods of diagnosis of enamel caries.
5. Treatment methods of enamel caries.

### **Question 1. Composition of enamel caries.**

Enamel is the most mineralized tissue of the body, forming a very hard, thin, translucent layer of calcified tissue that covers the entire anatomic crown of the tooth. Enamel is so hard because it is composed primarily of inorganic materials: 95% of enamel is calcium and phosphate ions combined to make up strong hydroxyapatite crystals. Hydroxyapatite crystals contain calcium and phosphate ions in the following proportions:  $\text{Ca}_{10}(\text{PO}_4)_6\text{OH}_2$ . Hydroxyapatite readily incorporates trace minerals into its crystal lattice. These ions can be negatively charged, such as fluoride or carbonate, or positively charged, such as sodium, zinc, strontium, or potassium. The concentrations of these trace minerals change the solubility of enamel. For example, the presence of fluoride in the crystal structure strengthens the structure and decreases solubility, while carbonate incorporation increases solubility. It has been found that hydroxyapatite crystals have more fluoride and less carbonate than crystals in the interior, making the outer surface less soluble than deeper layers of enamel.

Approximately 1% to 2% of enamel is made up of organic materials, particularly enamel-specific proteins called enamelin, which have a high affinity for binding hydroxyapatite crystals. Water makes up the remainder of enamel, accounting for about 4% of its composition.

The inorganic, organic, and water components of enamel are highly organized. Millions of carbonated hydroxyapatite crystals are arranged in long, thin structures called rods that are 4  $\mu\text{m}$  to 8  $\mu\text{m}$  in diameter<sup>2,4</sup>. Viewed in cross section, these rods appear as keyhole-shaped structures. It is estimated that the number of rods in a tooth ranges from 5 million in the lower lateral incisor to 12 million in the upper first molar. In general, rods extend at right angles from the dento-enamel junction (the junction between enamel and the layer below it called dentin) to the tooth surface. Surrounding each rod is a rod sheath made up of a protein matrix of enamelin. The area in between these rods is referred to as interrod enamel, or interrod cement. While it has the same crystal composition, crystal orientation is different, distinguishing rods from interrod enamel.

Some spaces exist where crystals do not form between rods. Typically called pores, they contribute to enamel's permeability, which allows fluid movement and diffusion to occur, but they also cause variations in density and hardness in the tooth, which can create spots that are more prone to demineralization - the loss of calcium and phosphate ions - when the oral pH becomes too acidic.

So, Enamel is most highly mineralized biological hard tissue. Enamel consists of apatite crystallites which are oriented in structural layers known as enamel prisms. The enamel crystals are surrounded by water. Enamel comprises 98% mineral present in the form of hydroxyapatite. The hydroxyapatite in human enamel is not pure. The hydroxyapatite contains carbonate ions. The presence of carbonate ions makes the enamel structure much more soluble and less resistant to acid dissolution. The hydroxyapatite is often described as a calcium-deficient carbonate hydroxyapatite  $\text{Ca}_{10-x}(\text{PO}_4)_6-x(\text{CO}_3)x(\text{OH})_2$ . A crystal structure resulting from the replacement of hydroxyl ions ( $\text{OH}^-$ ) in the hydroxyapatite structure with fluoride ions ( $\text{F}^-$ ) - Fluorapatite  $\text{Ca}_{10}(\text{PO}_4)_6\text{F}_2$ . Fluorhydroxyapatite is stronger and more acid resistant than hydroxyapatite.

### **Question 2. Histopathology of enamel caries.**

Caries process in enamel progresses through following stages.

**A. Early submicroscopic lesion.** There is prominence of Transverse striation of enamel and incremental striae of Retzius.

**B. Nonbacterial enamel destruction.** As caries advances, triangle or cone-shaped lesion appears the apex at A.D.J.

**C. Cavity formation and Bacterial invasion of enamel.**

Before complete disintegration of enamel (stage of white spot) several zones can be identified:

1. *Stage 1.* Translucent zone, characterized by presence of more pores, 1% of enamel surface (normal porosity is 0.1%) initial demineralization at the microscopical level. Formation of the translucent zone. No clinical or radiographical signs.

2. *Stage 2.* Dark zone, as a result of demineralization, presence of 2-4% pores. Further demineralization. Spread of the translucent zone. Initial redeposition of minerals within the defect. No clinical or radiographical signs.

3. *Stage 3.* Body lesion, Area of great demineralization and large pores, presence of 5-25% pores. Formation of the classical structure of the lesion Increased porosity in the body of the lesion clinically on the dry surface layer remains intact.



4. *Stage 4.* The surface zone, relatively unaffected as it has greater resistance due to a greater mineralization and presence of fluoride. Progression of the lesion towards the enamel dentin junction

### **Question 3. Clinical appearance of enamel caries**

#### ***ENAMEL CARIES (ICD-10)***

**K02.0** Caries limited to enamel:

- Enamel caries.
- Initial caries.

A non cavitated caries is the first clinically notable sign of the disease. Demineralization of hard dental tissues has reached the level when it can be seen with the naked eye but without a visible breakdown of dental enamel.

Under the microscope a variety of different irregularities can be observed:

- Destroyed perikymata pattern.
- Minor cracks and fractures.
- Microcavities.
- Scratches.

Histologically it appears as a wedge shaped defect with the base at enamel surface. The shape of the non cavitated lesion is determined by the distribution of the microbial deposits.

*White spot lesion.* The white spot lesion is the first visible evidence of a caries in the enamel, characterized by demineralized lesion underneath an intact surface. The lesion is caused by the accumulation of plaque and bacteria, developing among young adolescents with insufficient oral hygiene. The increased pore volume inside the demineralized lesion body leads to a different refraction index from the sound enamel. An inactive white spot lesion might act as arrested dental caries and impair only the esthetic appearance by displaying a milky white color from its interior opacity.

#### ***INITIAL CARIES***

Typical location. Approximal surfaces involve an interdental facet area toward the gingival margin, possible extensions buccally and lingually.

Occlusal surfaces natural pits and fissures are the most vulnerable sites. The process starts in the deepest parts of fissures, depending on tooth specific anatomy. Smooth surfaces along the gingival margin

Patient complains. No pain. Aesthetical discomfort especially in anterior teeth

#### **Clinical appearance**

##### **Active lesion:**

- Whitish, Opaque, Chalky.
- On smooth surfaces, close to gingival margin.
- Covered by plaque.

##### **Inactive lesion:**

- Whitish, Yellowish, brownish.
- Glossy and shiny.
- On smooth surfaces with small distance from gingival margin.
- Clean from plaque.

**Diagnosis:**

1. Gentle probing:
  - Active lesion feels rough.
  - Inactive feels smooth.
2. Dye method.
3. Radiograph detection conical shape in enamel, sometimes involve dentin.

**Differential diagnosis:** enamel hypoplasia, dental fluorosis.

### ***SUPERFICIAL CARIES***

**Patient complains**

- Short pain as reaction to sweets or thermal agents.
- Aesthetical discomfort especially in anterior teeth.

**Clinical appearance**

**Active lesion:**

- Whitish, Opaque, Chalky.
- On smooth surfaces shallow defect close to gingival margin.
- Covered by plaque.
- In fissures local enamel defect.

**Inactive lesion:**

- Whitish, Yellowish, brownish.
- Glossy and shiny.
- On smooth surfaces with small distance from gingival margin.
- Clean from plaque.
- On occlusal surfaces localized enamel defect.

**Diagnosis:**

1. Gentle probing:
  - Active lesion feels rough.
  - Inactive feels smooth and hard.
2. Radiograph detection conical shape in enamel, EDJ and outer dentin involve too.
3. FOTI for approximal surfaces a shadow in the area of a carious demineralization.
4. Electrometrical test show normal tooth vitality.

**Differential diagnosis:** enamel hypoplasia, dental fluorosis, usura cervicalis, dental erosion.

**Question 4. Main and additional methods of diagnostics of enamel caries**

The main methods of diagnosis:

**1. Questioning:**

- Complaints.
- Patient history.
- Medical history.

**2. Examination:**

- Probing.
- Drying.

**3. Visual assessment:**

- Reveals enamel caries (spot, cavity).
- Determines lesion activity.

**Caries Lesion Diagnosis**

*Visual and tactile methods* typically go hand in hand, because most dentists use dental probes and other tools to examine teeth during the clinical examination.

*Tools* - dental mirror, dental probe. Other tools used in visual–tactile examination may include magnifying devices to look at teeth, or orthodontic elastic separators to separate teeth over the course of 2 to 3 days for a closer look between teeth prone to caries lesions.

The traditional method of detecting caries signs is by visual inspection of dental surfaces, with the aid of a bright light and dental mirror if necessary to see teeth from all angles. Reflecting light onto the mouth mirror also can be done to search for dark shadows that could indicate dentin lesions.

The first visual indication of caries in enamel is generally a small white lesion on smooth surfaces or light to dark brown lesion in pits or fissures where demineralization has occurred under the dental plaque.

Careful visual inspection of well-dried tooth surfaces is useful in detecting discolored and demineralized areas of enamel and cementum. Incipient enamel caries lesions look whiter than the surrounding sound enamel because of the strong scattering of light within the lesion. Use of magnification can be especially helpful in assessing the integrity of the tooth surface.

The basis of visual inspection of caries is based upon the phenomenon of light scattering. Sound enamel is comprised of modified hydroxyapatite crystals that are densely packed, producing an almost transparent structure. The colour of teeth, for example, is strongly influenced by the underlying dentin shade. When enamel is disrupted, for example in the presence of demineralisation, the penetrating photons of light are scattered (i.e. they change direction, although do not lose energy) which results in an optical disruption. In normal, visible light, this appears as a ‘whiter’ area—the so called white spot. This appearance is enhanced if the lesion is dried; the water is removed from the porous lesion. Water has a similar refractive index to

enamel, but when it is removed, and replaced by air, which has a much lower refractive index than enamel, the lesion is shown more clearly. This demonstrates the importance of ensuring the clinical caries examinations are undertaken on clean, dry teeth.

Table 25. **Drying**

<i>Visual assessment</i>	<i>Histopathologic feature</i>
Enamel optical properties do not change after drying over 5 second	Lack of enamel demineralization
Enamel opacity or discoloration are not visible on the wet surface, but clearly visible after drying	Demineralization of the upper third of enamel
Enamel opacity or discoloration are clearly visible without drying	Entire demineralization of enamel and upper third of dentine

### **Lesion depth assessment.**

Ekstrand et al (1997) presented a visual ranked scoring system for lesion depth assessment that is still commonly used. Using no probe, they examined tooth surfaces to devise the following diagnostic levels:

- no or slight change in enamel translucency after 5 seconds of air-drying
- opacity or discoloration that is hardly visible on wet surfaces, but visible after 5 seconds of air drying
- opacity or discoloration that is visible without air-drying
- localized enamel breakdown with opaque or discolored enamel and/or grayish discoloration from underlying dentin
- cavitation in opaque or discolored enamel exposing dentin.

### **Recording both cavitated and non-cavitated lesions.**

Pitts and Fyffe (1988) devised the following diagnostic levels that are still used today, and devised this method with the help of a mouth mirror and probe:

- D1 (enamel lesion, no cavity).
- D2 (enamel lesions, cavity).
- D3 (dentin lesions, cavity).
- D4 (dentin lesions, cavity to the pulp).

### **Lesion activity assessment**

This is a newer diagnostic method developed by Nyvad et al in (1999). Diagnostic method focuses on the surface characteristics of lesions, namely activity as reflected in the surface texture of the lesion, and surface integrity, as indicated by the presence or absence of a cavity or microcavity in the surface. The rationale behind the method is that the surface characteristics of enamel change in response to changes in the biofilm covering the tooth surface. The diagnostic categories are as follows: active, non-cavitated;

active, cavitated; inactive, non-cavitated; inactive, cavitated; filling; filling with active caries; filling with inactive caries.

- Active, non-cavitated enamel caries lesions have a whitish/yellowish opaque surface, with a chalky or neon-white appearance, and the surface feels rough when a probe is moved across it.
- Inactive, non-cavitated lesions, on the other hand, are shiny and can vary in color from white, brown, or black, and will feel smooth with gentle probing.
- Active, cavitated lesions feel soft or leathery, while inactive, cavitated lesions are shiny and feel hard with probing.
- In general, active, non-cavitated lesions have a higher risk of progressing to a cavity than inactive, non-cavitated lesions, which have a higher risk of becoming a cavity than healthy surfaces.

**Diagnostic Criteria for Assessing Coronal Caries Lesion Activity  
according to International Caries Detection and Assessment System  
(ICDAS) (it was developed in 2002)**

The ICDAS lesion evaluation criteria serve as the basis for determining the stages of the caries process and lesion activity for the purpose of caries management. This is valid and reliable caries reporting system.

**Table 26. ICDAS II Criteria**

<b>Score</b>	<b>Criteria</b>
<b>0</b>	No or slight change in enamel translucency after prolonged air drying (five seconds)
<b>1</b>	First visual change in enamel (seen only after prolonged air drying or restricted to within the confines of a pit or fissure)
<b>2</b>	Distinct visual changes in enamel
<b>3</b>	Localized enamel breakdown in opaque or discoloured enamel (without visual signs of dentinal involvement)
<b>4</b>	Underlying dark shadow from dentin
<b>5</b>	Distinct cavity with visible dentin
<b>6</b>	Extensive distinct cavity with visible dentin (involving more than half of the surface)

For the purpose of caries management, individual tooth surfaces are categorized and described, based on an evaluation of each surface affected. For pits and fissures, the evaluation criteria are as follows:

**Additional methods of diagnosis**

**Fluorescence.** Laser fluorescence device (e.g. DIAGNOdent; Kavo GmbH, Bibberach, Germany) has been reported to be invalid in detecting carious lesions in occlusal surfaces

- Quantitative light-induced fluorescence (QLF, Inspektor, Amsterdam, The Netherlands).
- Fiber-optic transillumination (FOTI).
- Digitally imaging fiber-optic transillumination (DIFOTI).
- Electrical conductivity.
- Electrical impedance (CarieScan PRO™, CarieScan Ltd, Dundee, Scotland).
- Photothermal radiometry (Canary System™, Quantum Dental Technologies, Toronto).
- Radiologic Clinical Examination.

Fibre optic transillumination takes advantage of these optical properties of enamel and enhances them by using a high intensity white light that is presented through a small aperture in the form of a dental handpiece. Light is shone through the tooth and the scattering effect can be seen as shadows in enamel and dentine, with the device's strength the ability to help discriminate between early enamel and early dentine lesions. A further benefit of FOTI is that it can be used for the detection of caries on all surfaces; and is particularly useful at proximal lesions.

***Bitewing Radiography.*** The most commonly used radiographic method for detecting caries lesions is the bitewing technique. This technique is used for identifying carious lesions on the proximal surfaces that may be inaccessible to visual and tactile examination.

It is meant to find lesions that are hidden from a clinical visual examination, such as when a lesion is hidden by an adjacent tooth, as well to estimate depth of lesions. Another way in which bitewing radiography complements the visual–tactile examination is in the diagnosis of recurrent caries lesions.

To get the radiographic images, a central beam of X-rays is positioned to pass at right angles to the long axis of the tooth. If film is used, a beam-aiming device on the film holder guides the position, directing the beam at right angles to the film.

***Bitewing Radiography Diagnosis*** is that it is not invasive, and does not damage tooth structure like an incorrectly used dental probe might. Radiographs can also be filed and reexamined at a later date to compare with a more recent image to detect whether a lesion is progressing or not.

#### ***Limitations of Bitewing Radiography Diagnosis***

Besides concerns about low-dose radiation and variations in how images are interpreted by dentists, the main limitation is that the validity in diagnosing early lesions is rather low. Also, the bitewing radiograph cannot always distinguish between sound surfaces, those with initial caries activity and cavitated lesions, or non-carious demineralizations, so clinical inspection

is still needed to determine what is happening to the tooth. Bitewing radiographs also tend to underestimate the depths of lesions, so a lesion that appears confined to the inner enamel on an image is often actually in the dentin, and this can lead to insufficient or improper treatment.

However, digital radiography is replacing radiography based on film. It has been proven as accurate as traditional radiography for detecting caries, and it comes with additional advantages of using a lower radiation dose, being less time-consuming, and does not require wet chemicals in the processing of the image.

***Digital radiography*** – which is increasingly replacing bitewing radiography and that is as accurate as film for the detection of caries lesions.

***Digital image enhancement*** – which studies show can provide superior results to radiographs when enhanced correctly but takes a significant amount of technical skill.

Determination of the patient's caries risk based on past caries experience and current lifestyle factors is the logical first step in the diagnostic process. Properly exposed radiographs can detect proximal lesions that are only one third of the way through the enamel. Use of rectangular collimation and a film holding device can be beneficial by reducing the penumbra effect and providing a consistent exposure angle. Digital radiographic techniques may also improve the detection of early caries since the images acquired are digital and can be processed or analyzed to enhance diagnostic performance.

A key outcome of these efforts is the International Caries Classification and Management System (ICCMS™) a standardized method based on the best evidence currently available. This system, which is focused on improving long-term caries outcomes, combines history taking, clinical examination, risk assessment and personalized care planning at the individual patient level. A goal of the system is to develop a comprehensive care plan that incorporates:

- Preventing caries initiation (primary prevention).
- Preventive management of early caries (secondary prevention).
- Tooth preserving operative plan (minimally invasive).
- Review, monitoring and recall. This comprehensive care plan takes into account key risk factors for the individual patient, recommends inclusion of caries detection aids and lesion activity assessments and then lays out clear caries management strategies to obtain optimal results.

***Early caries detection and caries risk assessment***

The caries disease process is dynamic and multi-factorial in nature. Caries risk is defined as ‘the probability of future caries disease development’. Disease development includes both primary disease (new carious lesions) and secondary disease (lesion progression or reactivated

carious lesions). Risk assessment for such a dynamic disease is complex as it is only able to provide a snapshot at that particular moment and risk factors may change over time. Most importantly, for assessing lesion activity accurately in one session, using a combination of indicators (visual appearance, location, tactile sensation and gingival health) might still provide the best way to determine lesion activity.

The risk should be documented in a patient's chart and be used to influence a treatment plan.' One of the tools that assist clinicians worldwide in motivating patients is the 'Cariogram', an interactive validated program for patient motivation. This informative program illustrates caries risk in an instructive but simple graphical way, including the interaction between the various caries related factors. The Cariogram demonstrates the 'chance to avoid new carious lesion development' in the near future and to what extent the various factors will affect this chance. The software is available in 13 languages and can be downloaded as 'shareware'.

Caries-risk assessment is usually described at the level of the individual patient. It provides information needed to determine the most appropriate management decision for an individual patient. Risk prediction in a group is also pursued to enhance healthcare efficacy by focusing on those individuals with the largest risk, thus aiming to prevent or reduce a disease burden in the near future. This provides the oral healthcare professional with both individualized and population-based strategies for improving oral health.

The caries risk prediction may guide the best use of available funds to support preventive caries management, While the dental profession needs to embrace a more primary preventive approach to caries management based on common risk factors, secondary prevention and management will continue to focus on patient-centered caries management, including both preventive and minimally invasive tissue-preserving operative interventions

### **Question 5. Treatment methods of enamel caries.**

Therapeutics to promote the remineralization process. Remineralisation of enamel and dentine carious lesions.

**Fluoride. Mechanisms of action of fluoride in enamel.** The presence of fluoride during the remineralisation/demineralisation cycle leads to its incorporation into the crystalline structure of the carbonated hydroxyapatite, which not only decreases crystal solubility, but also increases the precipitation rate of enamel mineral in the presence of calcium and phosphate due to the lower solubility of fluorapatite. The fluoride decreases enamel solubility in two ways: (1) the fluoride ion is more stable in the crystal lattice than the hydrogen ion and (2) it interacts with the calcium ions on the crystal surface, interacting closely and binding strongly.



The effect and penetration of fluoride into the biofilm on the tooth surface is dependent on the type of fluoridated product and the time of exposure. When a clinical biofilm was exposed to 1,000 ppm (0.22%) sodium fluoride solution, exposure of up to 120 seconds increased plaque surface fluoride concentrations only, while 30-minute exposure allowed penetration of more than 1,000 ppm (0.22%) fluoride up to 900  $\mu\text{m}$  into the plaque.

Fluoride therapy has been the main caries preventive strategy since the introduction of water fluoridation schemes several decades ago. Fluoride is the most available and commonly used remineralization agent. Fluoride salts can be commonly found in drinking water, toothpastes and mouth rinses which are available to majority of the population in the world. However, it has been pointed out that, even with such a high availability, an increase in caries-free population reached a plateau in 1990s, and there were still at least 60% of teenagers around that time, and most likely still today, had observable dental decay. But the action of fluoride is limited by the amount of calcium derived from saliva, without extrinsic bioavailable sources of calcium and phosphate. Increased concentrations of calcium would also increase the retention of fluoride in the plaque biofilm by increasing calcium-bridging. Therefore, for remineralisation to occur during increased caries risk, an increase in bioavailable calcium and phosphate is fundamental to improving the effectiveness of the agent. Increased calcium and phosphate can be stabilised by macromolecules inherent in the saliva and plaque.

Thus the anti-caries effect of traditional fluoride therapy is still limited.

Many clinical studies have point out that fluoride therapy alone is not enough to overcome high caries challenges. Its retention in oral cavity is also another challenge.

Remineralizing products based on calcium phosphate remineralization systems. The technology which involves casein phosphopeptide stabilized amorphous calcium phosphate or casein phosphopeptide-amorphous calcium phosphate complexes (CPP-ACP).

The casein phosphopeptides (CPP) are derived from casein by tryptic digestion. In 1987, Reynolds found that CPPs were incorporated into the intra-oral appliance plaque and were associated with a substantial increase in the plaque's content of calcium and phosphate. Casein phosphopeptides (CPP) have the ability to stabilize high concentrations of calcium and phosphate in metastable solution, CPPs have a marked ability to stabilize calcium phosphate ions in solution and to form a amorphous calcium phosphate (ACP) complex, referred to as CPP-ACP. CPP-ACP binds strongly to hydroxyapatite and can diffuse and retained in dental plaque, therefore is able to buffer acid and substantially raise the level of calcium phosphate in plaque or close proximity to the tooth surface, and thus inhibits enamel demineralization and enhances remineralization. Stable and highly

soluble CPP-ACP has been trademarked as Recaldent™ and has now been commercialized in sugar-free gum and mints and in dental professional products (Tooth Mousse™).

Several randomized clinical trials (RCT) have shown that CPP-ACP added to sugar-free chewing gums, tooth paste or dental cream increased enamel subsurface remineralization. These RCT results suggested both a short-term remineralization effect of CPP-ACP and a caries-preventing effect for long-term clinical CPP-ACP use. Besides ACP, CPP also stabilize calcium fluoride phosphate (ACFP) and forming CPPACFP. In this case, calcium and phosphate ions co-localize at the tooth surface with fluoride ion, therefore increases the degree of saturation with respect to fluorapatite and promoting remineralization of enamel with fluorapatite.

A dicalcium phosphate anhydrous (DCPA) nanocomposite was developed by Xu, et al. as a restoration material that can slowly release high levels of  $\text{CaPO}_4$  requisite for remineralization.

The technology is an unstabilized amorphous calcium phosphate (ACP, Enamelon™).

The technology is a bioactive glass containing calcium sodium phosphosilicate (NovaMin™). Since all systems rely on calcium and phosphate compounds, their effect is mainly based on an enhancement of the natural capacity of saliva to remineralize mineral loss.

### **Techniques for Minimally Invasive Treatment**

The minimally invasive treatment options for dental demineralization or early caries include the following:

- 1) Treatment with topical fluoride and/or other remineralizing agents with repeated applications;
- 2) Surgical removal of demineralized enamel and placement of resin bonded restorative material;
- 3) Use of microinvasive infiltration resin;

Caries infiltration has been proposed as an alternative for management of non-cavitated enamel carious lesions on approximal and buccal surfaces.

The resin infiltration technique prevents further progression of the lesion using a low-viscosity resin with a high penetration coefficient, filling the enamel intercrystalline spaces. This technique has been reported to remove the whitish opaque color thereby changing the color and translucency of the white lesion. As such, the infiltrant creates a diffusion barrier for hydrogen ions preventing lesion progression.

Manufacturer's directions for the use of the infiltrant Icon® (DMG) are for treatment of proximal carious lesions extending no deeper than the outer third of the dentin layer radiographically, with demineralized "white spots" on the facial surfaces of teeth without visible cavitation.

On the enamel surface, careful application of 15% hydrochloric acid gel (ICONetch; DMG, Hamburg, Germany) is performed for 120 seconds to remove the surface layer less than 30 to 40  $\mu\text{m}$ . The acid gel was suctioned and washed away thoroughly. The lesions are desiccated using ethanol (ICON-Dry; DMG) for 30 seconds. After thorough desiccation with ethanol, an infiltrant resin (ICONinfiltrant; DMG) is placed on the tooth surface for 3 minutes for inside penetration. Excessive resin is wiped away from the surface and the proximal spaces. Light polymerizing is performed for 40 seconds. Applying the infiltrant resin was repeated to compensate for the shrinkage after polymerization. The tooth surface was polished with polishing discs.

**Pits and fissure sealants.** Pits and fissures of permanent molars are particularly prone to carious lesion development during and after tooth eruption. The morphology of pits and fissures has been reported to be one of the main caries risk factors, with molars being more frequently affected than premolars. Sealing aims to modify patent pits and fissures into smooth surfaces that are protected from bacterial colonization and exposure to fermentable substrate and can be cleaned easily. The strategy is effective not only as a preventive measure, but also in arresting non-cavitated enamel carious lesions in pits and fissures. The superiority of pit and fissure sealants over fluoride varnish application in the prevention of occlusal carious lesions has been reported.

Resin composites and glass-ionomer cements are the dental materials generally used to seal pits and fissures.

**Chemicomechanical Removal of Caries.** Method of removing caries is the chemicomechanical method as by using this procedure there are very less chances of having pain. The use of local anaesthesia while preparing the cavity has been reduced a lot by this procedure and the chemicals that are used are mainly available in two forms i.e. liquid form (caridex) and in gel form (carisolv).

**Procedure of using Carisolv gel.** In this procedure of chemicomechanical method of removing caries there is requirement to maintain the proper moisture control and there should be no chances of contamination. Rubber dam can be used as this can help a lot while working with chemicomechanical method of removing caries. The main steps that are to be followed by the specialist during the procedure are.

- First the tooth is properly diagnosed that where the carious lesion is present and this can be done with taking the x-ray of the tooth.
- Now apply the rubber dam on the tooth and then take small amount of gel and apply on the carious lesion with the help of any hand instrument.
- After 30 seconds take a sharp instrument with scraping end and remove the carious part.

- Now apply more gel and then again scrap out the carious part.
- Repeat the procedure until there left no carious part.

**Treatment of white spot lesions.** For esthetic improvement of non-cavitated white spot lesions with remineralized surface, treatment may consist of tooth bleaching, microabrasion, composite resin bonding, prosthetic restoration or some combination depending on the severity of the lesion and its etiology.

Some of the recent advances in dentistry regarding the cavity preparation method is air abrasion.



*Figure 5. Air abrasion instrument*

In air abrasion there is make the use of narrow and very powerful beam and it contains the aluminium oxide particles. The particles in this have the capability of being abrading the surface against which they got hit during the procedure. It produces no noise, no vibrations and no heat like the other cutting instruments. Also the procedure of cutting tooth material with the help of this is less painful and there occurs no shattering of enamel and other structure of the tooth. Air abrasion is most important to use in cavity preparation as it saves most of the tooth structure and prevent the excess cutting of it.

**Treatment of Superficial caries.** Surgical removal of demineralized enamel and placement of resin bonded restorative material;

### **Tests to the topic**

#### **1. Are the first visual indication of caries in enamel?**

- a. Grey lesions.
- b. Green lesions.
- c. White lesions.
- d. Black lesions.

**2. What are the basic tools for detecting caries lesions of enamel by visual inspection?**

- a. Bright light and a dental mirror.
- b. An X-ray.
- c. Fiber-optic transillumination.
- d. Electrical current.

**3. What is the primary purpose of caries diagnosis?**

- a. To only detect cavitations.
- b. To help identify early signs of tooth demineralization in order to halt its progression.
- c. To only detect non-cavitated tooth lesions.
- d. To only prevent teeth from falling out.
- e. All of the above.

**4. Which of the following is true about using a dental probe?**

- a. Excessive probing can cause irreversible damage to the surface of a developing lesion.
- b. Gentle probing does not disrupt the surface integrity of non-cavitated lesions.
- c. The blunt side can be used to remove biofilm.
- d. All of the above.

**5. Which visual–tactile set of diagnostic criteria is considered the best choice for performing caries lesion diagnosis?**

- a. Recording cavitated lesions only.
- b. Recording cavitated and non-cavitated lesions only.
- c. Activity assessment.
- d. Assessing lesion depth.
- e. All of the above.

**6. How do moisture levels on the tooth surface affect the visibility of a lesion?**

- a. White spot lesions become more opaque (visible) in dried dental tissue.
- b. White spot lesions become less visible in dried dental tissue.
- c. White spot lesions look greenish in dried tissue.
- d. Moisture levels do not affect the visibility of a lesion.

**7. Which of the following is a benefit of bitewing radiography?**

- a. It is not invasive and does not damage tooth structure like an incorrectly used probe might.

- b. It allows accessibility to surfaces that may not be seen in the clinical visual–tactile examination.
- c. Radiographs can be filed and reexamined at a later date to detect whether a lesion is progressing or not.
- d. All of the above.

**8. What is the main limitation of bitewing radiography?**

- a. The validity in diagnosing early lesions is rather low.
- b. It does a bad job of detecting caries in children's teeth.
- c. It requires too much equipment.
- d. This technique is too expensive.

**9. List remineralization agent:**

- a. Fluoride.
- b. Calcium.
- c. Phosphate.
- d. Resin composite.

**10. How do increased concentrations of calcium influence on retention of fluoride in the plaque biofilm?**

- a. Increase.
- b. Decrease.
- c. Do not influence.

## **LESSON 10. DENTINE CARIES: CLINICAL MANIFESTATIONS, DIAGNOSTIC METHODS. ISOLATION LINERS**

The questions to be studied for the learning of the topic:

1. Structure and composition of dentin.
2. Histopathology of dentin caries.
3. Clinical appearance of dentin caries.
4. Main and additional methods of diagnosis of dentin caries.
5. Differential diagnosis of dentin caries.

### **Question1. Structure and composition of dentin.**

Dentin formation begins immediately before enamel formation. Odontoblasts generate an extracellular collagen matrix as they begin to move away from the adjacent ameloblasts. Mineralization of the collagen matrix, facilitated by modification of the collagen matrix by various noncollagenous proteins, gradually follows its secretion. The most recently formed layer of dentin is always on the pulpal surface. This unmineralized zone of dentin is immediately next to the cell bodies of odontoblasts and is called predentin. Dentin formation begins at areas subjacent to the cusp tip or incisal ridge and gradually spreads to the apex of the root. In contrast to enamel formation, dentin formation continues after tooth eruption and throughout the life of the pulp. The dentin forming the initial shape of the tooth is called primary dentin and is usually completed 3 years after tooth eruption (in the case of permanent teeth).

The dentinal tubules are small canals that extend through the entire width of dentin, from the pulp to the DEJ. Each tubule contains the cytoplasmic cell process (Tomes fiber) of an odontoblast and in is layer of peritubular dentin, which is much mineralized than the surrounding intertubular dentin.

The surface area of dentin is much larger at the DEJ or dentinocemental junction than it is on the pulp cavity side. Because odontoblasts form dentin while progressing inward toward pulp, the tubules are forced closer together. The number of tubules increases from 15,000 to 20,000/mm<sup>2</sup> at the DEJ to 45,000 to 65,000/mm<sup>2</sup> at the pulp. The lumen of the tubules also varies from the DEJ to the pulp surface. In coronal dentin the average diameter of tubules at the DEJ is 0,5 to 0.9  $\mu\text{m}$ , but this increases to 2 to 3  $\mu\text{m}$  near the pulp.

The course of the dentinal tubules is a slight S-curve in the tooth crown, but the tubules are straighter in the incisal ridges, cusps, and root areas. The ends of the tubules perpendicular to the DEJ. Along the tubules walls are small lateral openings called canaliculi. As the odontoblastic process proceeds from the cell in the pulp to the DEJ, lateral secondary branches extend into the canaliculi and can communicate with the lateral

extensions of adjacent odontoblastic processes. Near the DEJ, the tubules divide into several terminal branches, forming an intercommunicating and anastomosing network.

Dentin is significantly softer than enamel but harder than bone or cementum. The hardness of dentin averages one-fifth that of enamel, and its hardness near the DEJ is about three times greater than near the pulp. Dentin becomes harder with age, primarily as a result of increases in mineral content. Although dentin is a hard, mineralized tissue, it is flexible, with a modulus of elasticity of approximately 18 GPa. This flexibility helps support the more brittle, nonresilient enamel. Dentin is not as prone to fracture as is the enamel rod structure. The ultimate tensile strength of dentin is approximately 98 MPa, whereas the ultimate tensile strength of enamel is approximately 10 MPa. The compressive strength of dentin and enamel are approximately 297 and 384 MPa, respectively.

During tooth preparation, dentin usually is distinguished from enamel by color and opacity, reflectance, hardness, and sound. Dentin is normally yellow-white and slightly darker than enamel. In older patients, dentin is darker, and it can become brown or black when it has been exposed to oral fluids, old restorative materials, or slowly advancing caries. Dentin surfaces are more opaque and dull, being less reflective to light than similar enamel surfaces, which appear shiny. Dentin is softer than enamel and provides greater yield to the pressure of a sharp explorer tine, which tends to catch and hold in dentin.

Sensitivity is encountered whenever odontoblasts and their processes are stimulated during operative procedures, even though the pain receptor mechanism appears to be within the dentinal tubules near the pulp. Physical, thermal, chemical, bacterial, and traumatic stimuli are transmitted through the dentinal tubules, although the precise mechanism of the transmissive elements of sensation has not been conclusively established. The most accepted theory of pain transmission is the hydrodynamic theory, which accounts for pain transmission through rapid movements of fluid within the dentinal tubules. Because many tubules contain mechanoreceptor nerve endings near the pulp, small fluid movements in the tubules arising from cutting, drying, pressure changes, osmotic shifts, or changes in temperature account for most pain transmission.

Dentinal tubules are filled with dentinal fluid, a transudate of plasma. When enamel or cementum is removed during tooth preparation, the external seal of dentin is lost, allowing tubular fluid to move toward the cut surface. Pulpal fluid has a slight positive pressure that forces fluid outward toward any breach in the external seal. Permeability studies of dentin indicate that tubules are functionally much smaller than would be indicated by their measured microscopic dimensions as result of numerous constrictions along



their paths. Dentin permeability is not uniform throughout the tooth. Coronal dentin is much more permeable than root dentin. There also are differences within coronal dentin. Dentin permeability primarily depends on the remaining dentin thickness (i.e., length of the tubules) and the diameter of the tubules. Because the tubules are shorter, more numerous, and larger in diameter closer to the pulp, deep dentin is a less effective pulpal barrier compared with superficial dentin.

### **Question 2. Histopathology of dentin caries.**

Caries in enamel is clearly a dynamic process, and this tissue does not contain cells and therefore is incapable of reacting in a vital manner. As soon as the process has reached dentin there is an immediate vital response by the odontoblasts and their processes within the dentinal tubules which is assumed as defense reaction. After reaching enamel dentin junction (EDJ), caries spreads laterally along the junction of least resistance and therefore undermines sound enamel. The established occlusal dentinal lesion is conical in shape with its basis on the EDJ and its apex directed towards the pulp. Continuous irritation of odontoblasts processes by acids diffusing through the porous enamel or even by bacteria themselves at the stage of enamel destruction activates the pulp/dentin organ in such a way that a region of reactionary or reparative irregular dentin begins to form from the pulpal side.

Dentin contains much less mineral and possesses microscopic tubules that provide a pathway for the ingress of bacteria and egress of minerals. The DEJ has the least resistance to caries attack and allows rapid lateral spreading when caries has penetrated the enamel. Because of these characteristics, dentinal caries is V-shaped in cross-section with a wide base at the DEJ and the apex directed pulpally. Caries advances more rapidly in dentin than in enamel because dentin provides much less resistance to acid attack owing to less mineralized content. Caries produces a variety of responses in dentin, including pain, sensitivity, demineralization, and remineralization.

Often, pain is not reported even when caries invades dentin except when deep lesions bring the bacterial infection close to the pulp. Episodes of short-duration pain may be felt occasionally during earlier stages of dentin caries. The pain is caused by stimulation of pulp tissue by the movement of fluid through the dentinal tubules that have been opened to the oral environment by cavitation. When bacterial invasion of the dentin is close to the pulp, toxins and possibly a few bacteria enter the pulp, resulting in inflammation of the pulpal tissues and, thus, pulpal pain.

The pulp-dentin complex reacts to caries attacks by attempting to initiate remineralization and blocking off the open tubules. These reactions result from odontoblastic activity and the physical process of demineralization and remineralization. Three levels of dentinal reaction to

caries can be recognized: (1) reaction to a long-term, low-level acid demineralization associated with a slowly advancing lesion; (2) reaction to a moderate-intensity attack; and (3) reaction to severe, rapidly advancing caries characterized by very high acid levels.

Dentin can react defensively (by repair) to low-intensity and moderate-intensity caries attacks as long as the pulp remains vital and has an adequate blood circulation.

In slowly advancing caries, a vital pulp can repair demineralized dentin by remineralization of the intertubular dentin and by apposition of peritubular dentin. Early stages of caries mild caries attacks produce long-term, low-level acid demineralization of dentin. Direct exposure of the pulp tissue to microorganisms is not a prerequisite for an inflammatory response. Toxins and other metabolic products, especially hydrogen ion, can penetrate via the dentinal tubules to the pulp. Even when the lesion is limited to enamel, the pulp can be shown to respond with inflammatory cells. Dentin responds to the stimulus of its first caries demineralization episode by deposition of crystalline material in the lumen of the tubules and the intertubular dentin of affected dentin in front of the advancing infected dentin portion of the lesion. Hypermineralized areas may be seen on radiographs as zones of increased radiopacity (often S-shaped following the course of the tubules) ahead of the advancing, infected portion of the lesion. This repair occurs only if the tooth pulp is vital.

Dentin that has more mineral content than normal dentin is termed sclerotic dentin. Sclerotic dentin formation occurs ahead of the demineralization front of a slowly advancing lesion and may be seen under an old restoration. Sclerotic dentin is usually shiny and darker in color but feels hard to the explorer tip. By contrast, normal, freshly cut dentin lacks a shiny, reflective surface and allows some penetration from a sharp explorer tip. The apparent function of sclerotic dentin is to wall off a lesion by blocking (sealing) the tubules. The permeability of sclerotic dentin is greatly reduced compared with normal dentin because of the decrease in the tubule lumen diameter.

Crystalline precipitates form in the lumen of the dentinal tubules in the advancing front of a demineralization zone (affected dentin). When these affected tubules become completely occluded by the mineral precipitate, they appear clear when a section of the tooth is evaluated. This portion of dentin has been termed affected zone of dentin and is the result of mineral loss in the intertubular dentin and precipitation of this mineral in the tubule lumen. Consequently, affected dentin is softer than normal dentin.

The second level of dentinal response is to moderate-intensity irritants. More intense caries activity results in bacterial invasion of dentin. Infected dentin contains a wide variety of pathogenic materials or irritants, including

high acid levels, hydrolytic enzymes, bacteria, and bacterial cellular debris. These materials can cause the degeneration and death of odontoblasts and their tubular extensions below the lesion and a mild inflammation of the pulp. The pulp may be irritated sufficiently from high acid levels or bacterial enzyme production to cause the formation (from undifferentiated mesenchymal cells) of replacement odontoblasts (secondary odontoblasts). These cells produce reparative dentin (reactionary dentin) on the affected portion of the pulp chamber wall. This dentin is different from the normal dentinal apposition that occurs throughout the life of the tooth by primary (original) odontoblasts. The structure of reparative dentin varies from well-organized tubular dentin (less often) to very irregular atubular dentin (more often), depending on the severity of the stimulus. Reparative dentin is an effective barrier to diffusion of material through the tubules and is an important step in the repair of dentin. Severe stimuli also can result in the formation within the pulp chamber of unattached dentin, termed pulp stones, in addition to reparative dentin.

The success of dentinal reparative responses, either by remineralization of intertubular dentin and apposition of peritubular dentin or by reparative dentin, depends on the severity of the caries attack and the ability of the pulp to respond. The pulpal blood supply may be the most important limiting factor to the pulpal responses.

The third level of dentinal response is to severe irritation. Acute, rapidly advancing caries with high levels of acid production overpowers dentinal defenses and results in infection, abscess, and death of the pulp. Compared with other oral tissues, the pulp is poorly tolerant of inflammation. Small, localized infections in the pulp produce an inflammatory response involving capillary dilation, local edema, and stagnation of blood flow. Because the pulp is contained in a sealed chamber, and its blood is supplied through narrow root canals, any stagnation of blood flow can result in local anoxia and necrosis. The local necrosis leads to more inflammation, edema, and stagnation of blood flow in the immediately adjacent pulp tissue, which becomes necrotic in a cascading process that rapidly spreads to involve the entire pulp.

Maintenance of pulp vitality depends on the adequacy of pulpal blood supply. Recently erupted teeth with large pulp chambers and short, wide canals with large apical foramina have a much more favorable prognosis for surviving pulpal inflammation than fully formed teeth with small pulp chambers and small apical foramina.

**Zone 1: normal dentin.** The deepest area is normal dentin, which has tubules with odontoblastic processes that are smooth, and no crystals are present in the lumens. The intertubular dentin has normal cross-banded collagen and normal dense apatite crystals. No bacteria are present in the

tubules. Stimulation of dentin (e.g., by osmotic gradient [from applied sucrose or salt], a bur, a dragging instrument, or desiccation from heat or air) produces a sharp pain.

**Zone 2: affected dentin.** Also called inner carious dentin, affected dentin is a zone of demineralization of intertubular dentin and of initial formation of fine crystals in the tubule lumen at the advancing front. Damage to the odontoblastic process is evident. Affected dentin is softer than normal dentin and shows loss of mineral from intertubular dentin and many large crystals in the lumen of the dentinal tubules. Stimulation of affected dentin produces pain. Although organic acids attack the mineral and organic contents of dentin, the collagen cross-linking remains intact in this zone. The intact collagen can serve as a template for remineralization of intertubular dentin, and this region remains capable of self-repair, provided that the pulp remains vital. The affected dentin zone can also be subclassified in three sub-zones: (1) subtransparent dentin (2) transparent dentin (3) and turbid dentin.

**Zone 3: infected dentin.** Also called outer carious dentin, this is the outermost carious layer, the layer, that the clinician would encounter first when opening a lesion. The infected dentin is the zone of bacterial invasion and is marked by widening and distortion of the dentinal tubules, which are filled with bacteria. Little mineral is present, and the collagen in this zone is irreversibly denatured. The dentin in this zone does not self-repair. This zone cannot be remineralized, and its removal is essential to sound, successful restorative procedures and the prevention of spreading the infection.

In slowly advancing lesions, it is expedient to remove softened dentin until the readily identifiable zone of sclerotic dentin is reached. In rapidly advancing lesions little clinical evidence (as determined by texture or color change) exists to indicate the extent of infected dentin.

### **Question 3. Clinical appearance of dentin caries.**

#### **Typical location:**

- Approximal surfaces a defect above the gingival margin, the ridge may be broken and the cavity extends to the occlusal surface.
- Occlusal surfaces a defect located in the pits and fissures and involving surrounding enamel.
- Smooth surfaces above the gingival margin.

#### ***Caries media***

#### **Patient complains:**

*Active lesion.* Short pain as reaction to sweets, sometimes there is the pain on thermal agents.

*Inactive lesion.* There are not complains.

### **Clinical appearance**

*Active lesion.* A cavity full of soft demineralized dentin. Light brown and dull.

*Inactive lesion.* Dark brown and shiny. On smooth surfaces with small distance from gingival margin.

**Diagnosis.** Gentle probing:

- Active lesion feels soft and sticking, sensitivity on probing.
- Inactive feels smooth and hard.

### ***Caries profunda***

- Short pain as reaction to sweets, thermal agents or mechanical pressure.
- High sensitivity on probing.
- Aesthetical discomfort especially in anterior teeth.

### **Clinical appearance**

*Active lesion.* A gross cavity full of soft demineralized dentin. Light brown and dull. On smooth surfaces shallow defect close to gingival margin.

*Inactive lesion.* Dark brown and shiny. On smooth surfaces with small distance from gingival margin.

**Diagnosis.** Gentle probing:

- Active lesion feels soft and sticking.
- Inactive feels smooth and hard.

Radiograph detection conical shape in enamel, EDJ and dentin at varying depth is usually involved too.

FOTI for approximal surfaces – shadow in the area of a carious demineralization

Electrometrical test show normal tooth vitality.

Term «**secondary caries**» defines the process of caries development which occurs after treatment of the primary caries lesion. Clinically it can be presented with all mentioned clinical manifestations, starting from a non cavitated lesion and extending further in enamel or dentin.

The reasons for formation of secondary caries are:

- Marginal gap formation between the restoration and the tooth surface.
- Broken filling.
- Loss of a part of the filling.
- Initial caries development due to plaque accumulation around margins of the filling.

**Diagnosis.** Can be performed with the same methods as for other forms of caries but in this case the visual examination of dental surfaces should be supported with additional diagnostic tools (bite wing). The 100% seal of the restored surface cannot be guaranteed forever and as soon as the dentinal

microbiota will obtain contact with oral environment, the destructive process in dentin towards the pulp will continue.

The marginal gap around the restorations cannot be detected with the naked eye at the initial stage but the products of microbial metabolism (acids) on the surface can penetrate the minor microporosities that appear in the restored tooth surface.

**Hidden caries lesion** in some areas which are relatively well protected from mechanical pressure, the layer of clinically intact enamel can be maintained for a long time, so hiding an ongoing demineralization in dentin.

In rare cases, if soft dentin is exposed to attrition the affected area gradually turned in to a smooth and polished surface, and the caries process may become arrested.

#### **Typical location**

- Approximal surfaces — a defect above the gingival margin, the ridge may be broken and the cavity extends to the occlusal surface.
- Occlusal surfaces — a defect located in the pits and fissures and involving surrounding enamel.
- Smooth surfaces above the gingival margin

#### **Patient complaints**

- Short pain as reaction to sweets, thermal agents or mechanical pressure.
- High sensitivity on probing.
- Aesthetical discomfort especially in anterior teeth.

**Clinical appearance-** a gross cavity full of soft demineralized dentin.

1. *Active lesion.* Light brown and dull. On smooth surfaces — shallow defect close to gingival margin.

2. *Inactive Lesion.* Dark brown and shiny. On smooth surfaces with small distance from gingival margin.

#### **Question 4. Main and additional methods of diagnosis of dentin caries**

Caries detection methods. Oral assessment and assignment of risk of developing dental caries. Radiologic and clinical examination. Emerging diagnostic techniques:

- Fluorescence
- Fiber-optic transillumination (FOTI)
- Digitally imaging fiber-optic transillumination (DIFOTI)
- Electrical conductivity.

Table 27. **Methods of caries detection based on their underlying physical principles**

<i>Physical principle</i>	<i>Application in caries detection</i>
X-rays	Digital subtraction radiography Digital image enhancement
Visible light	Fibre optic transillumination (FOTI) Quantitative light-induced fluorescence (QLF) Digital image fibre optic transillumination (DiFOTI)
Laser light	Laser fluorescence measurement (DiagnoDent)
Electrical current	Electrical conductance measurement (ECM) Electrical impedance measurement
Ultrasound	Ultrasonic caries detector

### **Clinical Examination for Dental Caries**

**Visual-tactile examination.** Visual changes of the dental structure resulting from the demineralization process can be visually observed during caries development, such as an increase in opacity and roughness of the enamel.

Visual examination has been widely used in dental clinics for detecting carious lesions on all surfaces. This method is based on the use of a dental mirror, a sharp probe and requires good lighting and a clean/dry tooth surface.

The examination is based primarily on subjective interpretation of surface characteristics, such as integrity, texture, translucency/opacity, location and color.

However, tactile examination of dental caries has been criticized because of the possibility of transferring cariogenic microorganisms from one site to another, leading to the fear of further spread of the disease in the same oral cavity. Moreover, use of an explorer can cause irreversible damages to the iatrogenic and demineralized tooth structure.

**Tooth separation** can be used as a method for examination of a suspicious area on the approximal surface. With this technique an orthodontic elastic separator can be applied for 2-3 days around the contact areas of approximal surfaces, facilitating the clinical and probing assessments. However, this method might create some discomfort and requires an extra visit. Studies have shown that tooth separation have detected more non-cavitated enamel lesions than visual-tactile examination without separation or bitewing examination.

The characteristics of active coronal lesions (not all characteristics need to be present to decide activity status) are as follows:

Table 28. **Characteristics of Active Coronal Lesions**

<i>ICDAS code</i>	<i>Characteristics of Lesion</i>	
	<i>Signs of Active Lesion</i>	<i>Signs of Inactive Lesion</i>
Initial to Moderate Stage Caries (1-4)	Surface of enamel is whitish/yellowish; opaque with loss of luster; feels rough when the tip of the probe is moved gently across the surface. Lesion is in a plaque stagnation area, i.e. in the entrance of pits and fissures, or near the gingiva, and in approximal surfaces below the contact point. Lesion was covered by thick plaque prior to cleaning.	Surface of enamel is whitish, brownish or black. Enamel may be shiny and feels hard and smooth when the tip of the probe is moved gently across the surface. For smooth surfaces, the caries lesion is typically located at some distance from the gingival margin. Lesion was not covered by thick plaque prior to cleaning.
Extensive Stage Caries (S-6)	Dentine feels soft or leathery on probing.	Dentine is shiny and hard on probing.

For the purpose of caries management, individual tooth surfaces are categorized and described, based on an evaluation of each surface affected. For pits and fissures, the evaluation criteria are as follows:

Table 29. **Pits and Fissures Evaluation Criteria**

<i>Tooth Surface Description</i>	<i>Evaluation Criteria</i>	<i>ICDAS Code</i>
Sound surfaces	No visible caries when viewed clean and dry. Non-carious white or brown marks on tooth surfaces must be differentiated from early caries lesions.	0
Initial stage caries	Characterized by the first visual change in enamel (seen only after prolonged air drying or restricted to the confines of a pit or fissure). <b>OR</b> A distinct visual change in enamel (seen on a wet or dry surface).	1
		2
Moderate stage caries	Characterized visually by either localized enamel breakdown (without visual signs of dentinal exposure). - Enamel breakdown is often viewed best when the tooth is air dried. <b>OR</b> An underlying dark shadow from dentin. - Shadowing from dentinal caries is often best seen with the tooth surface wet.	3
		4



Extensive stagecaries	Characterized by distinct cavitation exposing visible dentine. - Lesions exhibiting cavitation Involving less than half the tooth surface - Lesions involving half of the tooth surface or more	5
		6

For mesial and distal surfaces, the evaluation criteria are as follows:

**Table 30. Mesial and Distal Surfaces Evaluation Criteria**

<i><b>Tooth Surface Description</b></i>	<i><b>Evaluation Criteria</b></i>	<i><b>ICDAS Code</b></i>
Sound surfaces	No visible caries when viewed clean and dry. Non-carious white or brown marks on tooth surfaces must be differentiated from early caries lesions.	0
Initial stage caries	Characterized by the first visual change in enamel (seen only after prolonged air drying). <b>OR</b> A distinct visual change in enamel (seen on a wet or dry surface). - These lesions are usually seen directly from the lingual or buccal directions but may be viewed from the occlusal direction as a shadow confined to enamel.	1
		2
Moderate stage caries	Characterized visually by either localized enamel breakdown (without visual signs of dentinal exposure). - Enamel breakdown is often viewed best when the tooth is air dried. <b>OR</b> An underlying dark shadow from dentin. - Shadowing from dentinal caries is often best seen with the tooth surface wet.	3
		4
Extensive stagecaries	Characterized by distinct cavitation exposing visible dentine. - Lesions exhibiting cavitation Involving less than half the tooth surface - Lesions involving half of the tooth surface or more	5
		6

**Table 31. Buccal-lingual Smooth Surfaces Evaluation Criteria**

<i><b>Tooth Surface Description</b></i>	<i><b>Evaluation Criteria</b></i>	<i><b>ICDAS Code</b></i>
Sound surfaces	No visible caries when viewed clean and dry. Developmental defects like enamel hypoplasias, fluorosis, tooth wear (attrition, abrasion and erosion), and extrinsic or intrinsic stains should be recorded as sound in the absence of other signs of caries lesions as described below.	0
Initial stage caries	Characterized by the first visual change in enamel (seen only after prolonged air drying).	1

	<b>OR</b> A distinct visual change in enamel (seen on a wet or dry surface). - Initial stage lesions on free smooth surfaces are located in close proximity (In touch or within 1 mm) to the gingival margin or adjacent to orthodontic or prosthetic attachments on a tooth surface	2
Moderate stage caries	Characterized visually by either localized enamel breakdown (without visual signs of dentinal exposure). - Enamel breakdown is often viewed best when the tooth is air dried. <b>OR</b> An underlying dark shadow from dentin. - Shadowing from dentinal caries is often best seen with the tooth surface wet.	3
		4
Extensive stage caries	Characterized by distinct cavitation exposing visible dentine. - Lesions exhibiting cavitation involving less than half the tooth surface - Lesions involving half of the tooth surface or more	5
		6

### **Radiologic Clinical Examination for Dental Caries**

*The use of X-rays and radiographic films* promoted a significant jump in the direction of dental therapy, since it provided substantial contribution in obtaining the diagnosis. In addition, radiographic techniques have been modified to acquire optimum X-ray quality and to increase diagnostic possibilities, as for detecting caries lesions.

Radiography is the most common caries lesion detection aid. It is fundamentally based on the fact that as the caries progress proceeds, the mineral content of enamel and dentin decreases, resulting in a decrease in the attenuation of the X-ray beam as it passes through the teeth. This feature is recorded on the image receptor as an increase in radiographic density. Clinically, the detection of carious lesions is based on a combination of visual-tactile and radiographic examination.

Bitewing radiography has been used for the detection and evaluation of caries lesions depth, which are invisible or poorly visible for inspection. Thus, radiography is mainly used for the detection of carious lesions in approximal surfaces, but is also recommended as a supplement for occlusal caries detection. However, experiments have shown that, once an occlusal carious lesion is clearly visible on radiographs, histological examination shows that demineralization has extended to or beyond the middle third of the dentin.

Regarding the performance of bitewing radiography, studies have found that the X-rays show a high sensitivity (50-70%) to detect caries

lesions in dentin of both approximal and occlusal surfaces, compared to clinical visual detection. However, the validity of detecting enamel lesions is limited on the approximal surfaces and low for the occlusal surfaces. This difference can be explained by the fact that radiography is a 2-dimensional image of a 3-dimensional anatomy of the tooth structure.

Several criteria are used to classify the extent of carious lesions on radiographs, such as:

- (0) absence of radiolucency
- (1) radiolucency in the outer half of the enamel
- (2) radiolucency on the inner half of the enamel, which can extend up to the dentin-enamel junction (DEJ)
- (3) radiolucency in the outer half of the dentin
- (4) radiolucency in the inner half of the dentin toward the pulp chamber.

***Bite-wing (BW) films*** for proximal decay detection

#### **Directions in Caries Diagnosis**

***Occlusal caries.*** Caries researchers recommend against using a sharp explorer when examining pits and fissures. Histological evidence shows the explorer can disrupt incipient caries and bacteria can be moved from groove to groove by a sharp explorer. Reliance on a sharp explorer has the potential to underutilize other detection methods and leads to a false positive diagnosis of occlusal caries.

Visualization of pit and groove areas for discoloration under the enamel is essential for pit and fissure diagnosis. Teeth must be clean, dry, and well illuminated to be properly evaluated. With the greater exposure to fluorides and more frequent placement of sealants deep caries can exist without a visible indication. Radiographs, in the past considered of limited use in the detection of occlusal caries, have been shown to be valuable in detecting deep dentin caries beneath pit and fissures and sealants.

***Proximal caries.*** Proximal radiographs provide a good representation of caries in enamel and dentin. When exposed with consistent angulation radiographs can provide an indication of whether a lesion is progressing. Film holding devices are valuable in producing consistent radiographic exposures, and should be used whenever possible. Visualization of the tooth looking for clinical cavitation and marginal ridge discoloration is an important adjunct to radiographic examination. Transillumination of the tooth is a valuable adjunct when recent films are not available, but provides limited additional diagnostic yield beyond that afforded by a radiograph. An evaluation of risk is a required part of the prescription of a radiographic recall interval.

The radiographic examination recommendations are presented based. The recommendations are meant only as a guide and may be customized by

the dentist to satisfy the individual needs of the patient in a given situation. As an example, specific monitoring of an early carious lesion would be appropriate at other intervals.

As the patient's caries risk level increases, the need for timely clinical dental examination increases.

Using criteria based on the caries risk level helps to ensure that patients will not be exposed to unnecessary ionizing radiation and that the radiographs taken will have significant diagnostic value. Intervals for clinical examination are lengthened as the dentist has gathered information that indicates the caries risk is reduced. Monitoring of patient compliance with lifestyle changes and interventions

The detection of carious lesions has been primarily a visual process, based principally on clinical-tactile inspection and radiographic examination. Caries detection methods should be capable of detecting lesions at an early stage, when progression can be arrested or reserved, avoiding premature tooth treatment by restorations. However, none of the conventional methods fulfill this requirement and are highly subjective. The development of some alternative non-invasive detection methods, such as laser fluorescence devices (DIAGNOdent and DIAGNOdent pen), quantitative light-induced fluorescence (QLF), fluorescence camera (VistaProof), LED technology (Midwest Caries I.D.), fiber-optic transillumination (FOTI), digital imaging fiber-optic transillumination (DIFOTI) and electrical caries monitor (ECM), can offer objectives assessments, where traditional methods could be supplemented by quantitative measurements.

### Question 5. Differential diagnosis of dentin caries

Table 32. **Differential diagnosis pulpitis symptomatica, pulpitis asymptomatica, periodontitis asymptomatica.**

	<b>Caries profunda</b>	<b>Pulpitis symptomatic</b>	<b>Pulpitis asymptomatic</b>	<b>Periodontitis asymptomatic</b>
<b>State of the cavity</b>	Deep, full of soft discolored dentin, no pulp exposure.	Deep, full of soft discolored dentin, may be pulp exposure.	Deep, full of soft discolored dentin, may be pulp exposure.	Deep, full of soft discolored dentin, may be pulp exposure.
<b>Character of pain</b>	No irritation. No pain.	Prolonged pain, Self starting pain. Irritation induce pain.	Prolonged pain after irritation.	No pain.
<b>Reaction to thermal irritants (cold, heat)</b>	Short pain, Pain disappear after removal of agent.	Prolonged pain to cold. Doesn't stop after removal of agent. Irradiates	Prolonged throbbing (pain to cold or heat).	No reaction to thermal agents.

		to nerve.		
<b>Electro test</b>	Normal 10-20 $\mu$ a.	Lowered 20-40 $\mu$ a.	Lowered 20-40 $\mu$ a.	No reaction 100-200 $\mu$ a
<b>X-ray examination</b>	Focal radiolucency in dentin, Thin layer of dental bridge above pulp, No changes in periodontium.	Radiolucency in dentin extending to the pulp. No changes in periodontium.	Radiolucency in dentin extending to the pulp. Periodontal gap slightly widened.	Radiolucency involves the pulp. Pathological changes are present in periodontium.

### Tests to the topic

#### 1. What differentiates dentin from enamel?

- There are no significant differences.
- Enamel can repair and regenerate, while dentin cannot.
- Unlike enamel, dentin is living tissue with the ability for constant growth and repair, thanks to cells called odontoblasts that create new dentin.
- Dentin is harder than enamel.

#### 2. What is the basic dentin element?

- Dentinal tubule.
- Odontoblast.
- Enamel rod.
- Lamella.

#### 3. What is the average diameter of dentinal tubules at the DEJ?

- 0,5 to 0.9  $\mu$ m.
- 2 to 10  $\mu$ m.
- 100 to 200  $\mu$ m.
- 50 to 100  $\mu$ m.

#### 4. What is the average diameter of dentinal tubules near the pulp?

- 0,5 to 0.9  $\mu$ m.
- 2 to 10  $\mu$ m.
- 100 to 200  $\mu$ m.
- 2 to 3  $\mu$ m.

#### 5. Dentin includes the following parts:

- Peritubular dentin.
- Intertubular dentin.
- Dentinal tubules.

- d. Odontoblasts.

**6. What does the dentinal tubule contain?**

- a. Cytoplasmic cell process of an odontoblast.
- b. Dentinal fluid.
- c. Intertubular dentin.
- d. Pulp.

**7. What is the most accepted theory of pain transmission in the hard tissues of teeth?**

- a. Hydrodynamic theory.
- b. Neurological theory.
- c. Microbial theory.
- d. Receptor theory.

**8. What are the patient complaints at caries profunda?**

- a. Short pain as reaction to sweets, thermal agents or mechanical pressure.
- b. Extending pain.
- c. Pain when biting
- d. Lasting pain as reaction to thermal agents or mechanical pressure.

**9. What are the patient complaints at caries media?**

- a. Short pain as reaction to sweets and others chemical agents.
- b. Extending pain.
- c. Pain when biting.
- d. Lasting pain as reaction to thermal agents or mechanical pressure.

**10. What are clinical appearances of acute caries profunda?**

- a. A gross cavity full of soft demineralized dentin.
- b. High sensitivity on probing of cavity bottom.
- c. Walls of cavity are light brown and dull.
- d. Small enter of caries cavity.
- e. All of the above.

## LESSONS 11. DENTINE CARIES. TREATMENT METHODS. HEALING LINERS

The issues to be studied for the learning of the topic:

1. Principles of tooth preparation.
2. Instrument and Equipment for Tooth Preparation.
3. Stages of carious cavity preparation.
4. Pulp protection.
5. Classification of healing liners.
6. Treatment of deep caries lesions.

### **Question 1. Principles of tooth preparation.**

- To restore function.
- To prevent further spread of an active lesion which is not amenable to preventive measures.
- To preserve pulp vitality.
- To restore aesthetics.

***Preparation design.*** With caries prevalence declining, emphasis has changed from extension for prevention, to minimizing removal of tooth tissue. Tooth preparation should be based on the morphology of the carious lesion and the requirements of the restorative material being used.

#### General principles of tooth preparation:

- 1) Gain access to caries.
- 2) Remove all caries at ADJ (to prevent spread laterally).
- 3) Cut away all significantly unsupported enamel.
- 4) Extend margins so that they are accessible for instrumentation and cleaning.
- 5) Shape preparation so that remaining tooth tissue and restorative material will be able to withstand functional forces.
- 6) Shape preparation so that restoration will be retained, i. e. undercut for amalgam, none required for resin composite or bonded amalgams.
- 7) Check preparation margins are appropriate for the restorative material. Small areas of unsupported enamel may be left if a resin composite restoration is being placed.
- 8) Remove remaining caries unless indirect pulp cap to be carried out.
- 9) Wash and dry preparation.
- 10) While care must be exercised not to overcut a preparation, do not skimp on access so that caries removal is compromised by poor visibility.
- 11) Mark centric stops with articulating paper prior to tooth preparation and try to preserve if possible, or place the preparation margins past the occlusal contact areas.
- 12) Avoid crossing marginal ridges.

- 13) In removing caries a tactile appreciation of the hardness of dentine is important, therefore use slow-speed instruments or excavators.
- 14) The base of the preparation should not be flattened as this runs the risk of pulp exposure.
- 15) Unless caries dictates, margins should be supragingival.
- 16) All internal line angles should be rounded to internal stresses. Removing caries with a large diameter round bur automatically produces the desired shape.
- 17) In a proximal box, the margin should extend below the contact point because this is where the caries is!

***Approach to caries preparation.*** The old surgical approach to cavity design was adopted in the absence of adhesive techniques and on the basis of Black's principle of "extension for prevention," but this theory is no longer tenable.

The current availability of adhesive bioactive restorative materials makes it possible to maintain areas of tooth structure even though they appear to be undermined and weakened. Thus, the concept of geometric designs for cavities is no longer valid.

## **Question 2. Instrument and Equipment for Tooth Preparation.**

Removal and shaping of tooth structure are essential aspects of restorative dentistry.

***Rotary Power Cutting Equipment.*** Powered rotary cutting instruments, known as dental hand-pieces, are the most commonly used in contemporary dentistry. Two technologies are used today for dental handpieces. The air-driven handpiece was, for many years, the mainstay for cutting teeth in dentistry. The electric motor-driven handpiece is now becoming increasingly popular for use in all cutting applications in dentistry. The advantages of electric handpieces are that they are quieter than air-driven handpieces, they cut with high torque with very little stalling, they maintain high bur concentricity, and they offer high-precision cutting. Cutting with electric handpieces is smoother and more like milling, whereas cutting with the air-driven handpiece is more like chopping the tooth with the bur. Another advantage of electric handpieces is that they offer multiple attachment for the motor that can be used for different cutting applications such as denture adjustments and endodontic instrumentation. Some disadvantages of air-driven handpieces are that they create a loud, high-pitched noise. The torque and concentricity of the air turbines degrade in a relatively short period. More vibration and bur chatter are associated with air-driven handpieces.

### ***Rotary speed Ranges for different cutting applications***

The rotational speed of an instrument is measured in revolutions per minute (rpm). Three speed ranges are generally recognized: low or slow



speeds (<12.000 rpm), medium or intermediate speeds (12.000-200.000 rpm), and high or ultra-high speeds (>200.000 rpm). Most useful instruments are rotated at either low speed or high speed. Electric handpiece motors generate up to 200.000 rpm of rotation. This speed is significantly less than the 400.000 rpm generated by air-driven handpieces. However, the Electric handpiece motor has attachments with speed increase multipliers that can increase rotation in ratios of 5:1 or 4:1, which makes them effective in the same range as air-driven handpieces.

The crucial factor for some purposes is the surface speed of the instrument, that is, the velocity at which the edges of the cutting instrument pass across the surface being cut. This speed is proportional to the rotational speed and the diameter of the instrument, with large instruments having higher surface speeds at any given rate of rotation. Low-speed cutting is ineffective, is time-consuming, and requires a relatively heavy force application; this results in heat production at the operating site and produces vibrations of low frequency and amplitude.

Heat and vibration are the main sources of patient discomfort. At low speeds, burs have a tendency to roll out of the tooth preparation and mar the proximal margin or tooth surface. In addition, carbide burs do not last long because their brittle blades are easily broken at low speeds. Many of these disadvantages of low-speed operation do not apply when the objective is some procedure other than cutting tooth structure. The low-speed range is used for cleaning teeth, caries excavation, and finishing and polishing procedures. At low speeds, tactile sensation is better, and generally, overheating of cut surfaces is less likely. The availability of a low-speed option provides a valuable adjunct for many dental procedures.

At high speed, the surface speed needed for efficient cutting can be attained with smaller and more versatile cutting instruments. This speed is used for tooth preparation and removing old restorations. Other advantages are the following: (1) diamond and carbide cutting instruments remove tooth structure and with less pressure, vibration, and heat generation; (2) the number of rotary cutting instruments needed is reduced because smaller sizes are more universal in application; (3) the operator has better control and greater ease of operation; (4) instruments last longer; (5) patients are generally less apprehensive because annoying vibrations and operating are decreased; and (6) several teeth in the same arch can be treated at the same appointment (as they should be).

### ***Rotary Cutting Instruments. Common Design. Characteristics***

Despite the great variation among rotary cutting instrument they share certain design features. Each instrument consists of three parts: (1) shank, (2) neck, and (3) head. Each has its own function, influencing its design and the

materials used for its construction. The term shank has different meanings as applied to rotary instruments and to instruments.



Figure 6. Normal designation of three parts of rotary cutting instruments.

**Diamond instruments** consist of three parts: a metal blank, the powdered diamond abrasive, and a metallic bonding material that holds the diamond powder onto the blank. The blank in many ways resembles a bur without blades. It has the same essential parts: head, neck, and shank.

The shank dimensions, similar to those for bur shanks, depend on the intended handpiece. The neck is normally a tapered section of reduced diameter that connects the shank to the head, but for large disk-shaped or wheel-shaped instruments, it may not be reduced below the shank diameter. The head of the blank is undersized compared with the desired final dimensions of the instrument, but its size and shape determine the size and shape of the finished instrument. Dimensions of the head make allowance for a fairly uniform thickness of diamonds and bonding material on all sides. Some abrasive instruments are designed as a mandrel and a detachable head. This is much more practical for abrasive disks that have very short lifetimes.

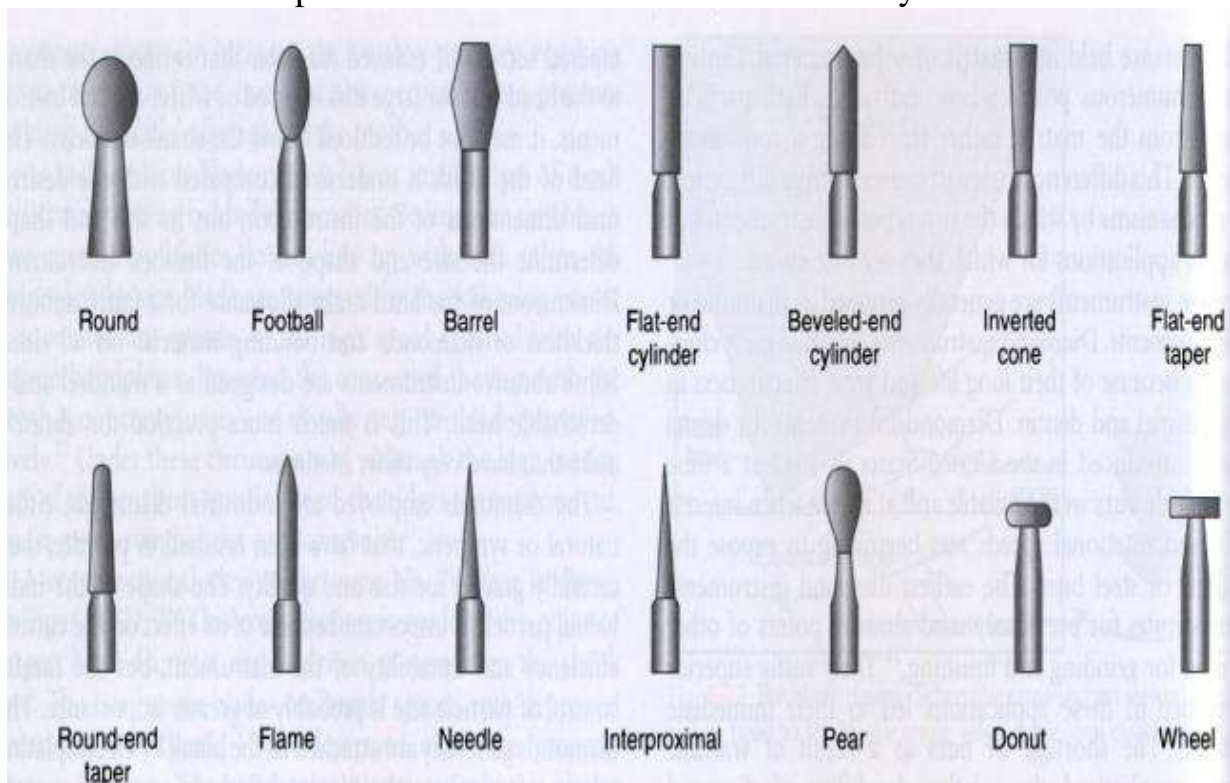


Figure 7. Characteristic shapes and designs for a range of diamond cutting instruments

**Head Shapes and Sizes.** Diamond instruments are available in a wide variety of shapes and in sizes that correspond to all except the smallest-diameter burs. The greatest difference lies in the diversity of other sizes and shapes in which diamond instruments are produced. Even with many subdivisions, the size range within each group is large compared with that found among the burs. More than 200 shapes and sizes of diamonds are currently marketed. It is essential to indicate the manufacturer when attempting to describe diamond instruments by catalogue number.

**Diamond Particle Factors.** The clinical performance of diamond abrasive instruments depends on the size, spacing, uniformity, exposure, and bonding of the diamond particles. Increased pressure causes the particles to dig into the surface more deeply, leaving deeper scratches and removing more tooth structure. Diamond particle size is commonly categorized as coarse (125-150  $\mu\text{m}$ ), medium (88-125  $\mu\text{m}$ ), fine (60-74  $\mu\text{m}$ ), and very fine (38-44  $\mu\text{m}$ ) for diamond preparation instruments. These ranges correspond to standard sieve sizes for separating particle sizes.

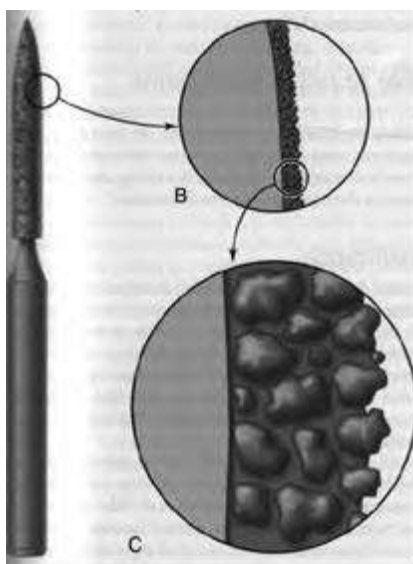


Figure 8. Diamond particles.

When using large particle sizes, the number of abrasive particles that can be placed on a given area of the head is decreased. For any given force that the operator applies, the pressure on each particle tip is greater. The resulting pressure also is increased if diamond particles are more widely spaced so that fewer are in contact with the surface at any one time. The final clinical performance of diamond instruments is strongly affected by the technique used to take advantage of the design factors for each instrument.

Diamond finishing instruments use even finer diamonds (10-38  $\mu\text{m}$ ) to produce relatively smooth surfaces for final finishing with diamond polishing

pastes. Surface finish less than 1  $\mu\text{m}$  are considered clinically smooth and can be routinely attained by using a series of progressively finer polishing steps.

### **Question 3. Stages of carious cavity preparation.**

1. Opening of cavity
2. Enlargement of cavity.
3. Necrectomy is a removal of the necrotic dental hard tissues from carious cavity.
4. Formation the cavity.
5. Smoothing the edges of enamel.

The use of modified cavity designs for the treatment of initial carious lesions can be justified on the grounds that, because no restorative material can adequately replace natural tooth structure for the long term, preservation of natural tooth structure is important. It is apparent that it is possible to remineralize and heal demineralized tooth structure to some degree. Therefore, neither enamel nor dentin should be removed simply because it has lost calcium and phosphate ions as a result of acid attack. The older surgical approach to cavity design was adopted in the absence of adhesive techniques and on the basis of Black's principle of "extension for prevention," but this theory is no longer tenable. The current availability of adhesive bioactive restorative materials makes it possible to maintain areas of tooth structure even though they appear to be undermined and weakened. Thus, the concept of geometric designs for prescribed cavities is no longer valid. The purpose of this article is to describe a series of simplified, modified cavity designs for small initial lesions; preservation of natural tooth structure is the principle objective of these designs.

### **Question 4. Pulp protection.**

#### **Pulpal Responses**

<i>Types of stimulus</i>	<i>Examples of stimulus</i>
Physical	Thermal, electrical
Mechanical	Handpiece, traumatic occlusion
Chemical	Acid from dental materials
Biologic	Bacteria from saliva

- 1) Heat generated by rotary instruments,
- 2) Some ingredients of various materials
- 3) Thermal changes conducted through restorative materials
- 4) Forces transmitted through materials to the dentin
- 5) Galvanic shock
- 6) The ingress of different products and bacteria through microleakage.

It has been generally accepted that the materials that were used to restore teeth posed a danger to the tooth and allowed for the occurrence of postoperative sensitivity. If this were true, then a barrier or protective layer needed to be placed on the tooth before the final restoration. This buffer would, in part, act to reduce or even eliminate postoperative sensitivity.

Over time we have come to learn that it is not the restorative material that causes problems, but bacteria and the by-products of bacteria. These bacteria, present in the oral cavity, enter the tooth at the margin of the restoration through capillary action of oral fluids. This is referred to as microleakage. Others have defined microleakage as 'the marginal permeability of bacterial, chemical, and molecular invasion at the interface between the teeth and restorative material.

Liner and bases Intermediate supplementary restorative materials are materials that are placed between a restoration and the dentine with a primary function of protecting the pulp.

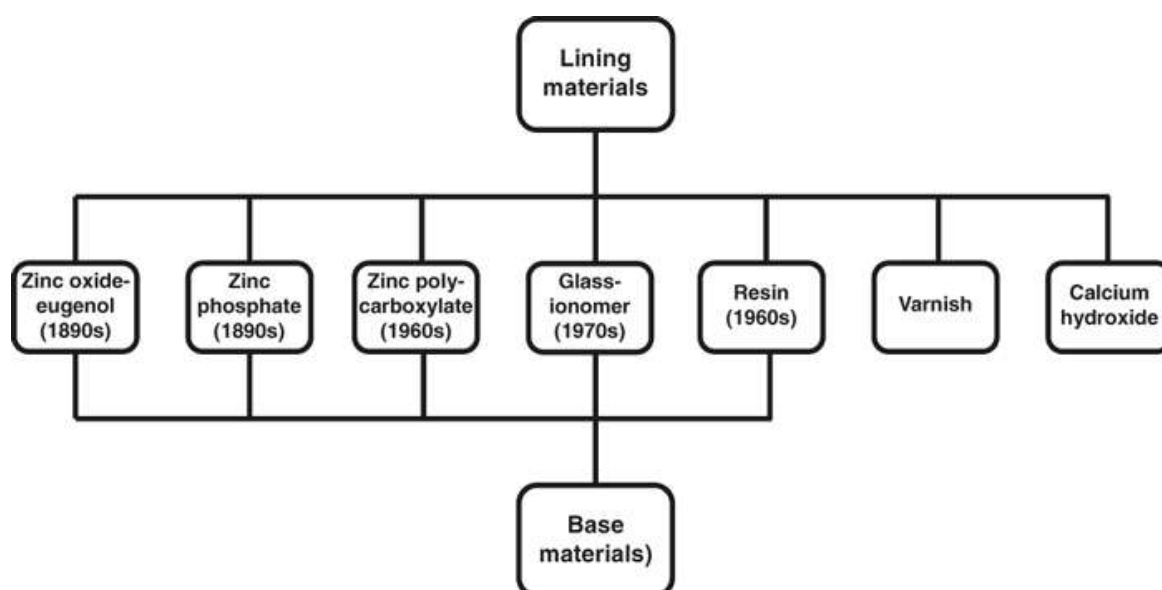
Liners include suspensions or dispersions of zinc oxide, calcium hydroxide, resin-modified glass ionomer. It can be applied to a tooth surface in a relatively thin film. Dental liners provide a thin barrier that protects the pulpal tissue from irritation caused by physical, mechanical, chemical and biologic element. Liners protect dentin from irritant agents from either the restorative materials or oral fluids. Bases are materials, most composites, that are used in thicker dimensions beneath restorations to provide for mechanical, chemical, thermal protection of the pulp.

Examples: phosphate, zinc oxide-eugenol, polycarboxylate cements and most common, some type of glass ionomer (usually an RMGI).

If the removal of infected dentin does not extend deeper than 1 to 2 mm from the initially prepared pulpal or axial wall, usually no liner is indicated.

If the excavation extends into or within 0.5 mm of the pulp, a calcium hydroxide liner usually is selected to stimulate reparative dentin (indirect pulp cap procedure).

**Materials which can be used as liners and bases,  
with approximatedate of introduction**



Many of these materials belong to the family of so-called water-based «cements».

**Table 33. Liners and Bases application in order of use**

<i>Type of restorative material</i>	<i>Shallow Preparation</i>	<i>Moderately Deep Restoration</i>	<i>Deep Restoration</i>
Amalgam	1.Dentin sealer 2.Bonding system	1.Base 2.Dentin sealer 3.Bonding system	1.Liner 2.Base 3.Dentin sealer 4.Bonding system
Composite resin	1.Bonding system	1. Bonding system	1.Liner 2.Bonding system
Gold inlays/onlays		1. Base	1.Liner 2.Base
Ceramic	1.Bonding system	1. Bonding system	1.Liner 2.Bonding system

**Question 5. Classification of healing cavity liners.**

**Calcium hydroxide.** Calcium hydroxide (CaOH) has two components: a base and a ‘catalyst’. The base is composed of calcium tungstate, tribasic calcium phosphate and zinc oxide. The catalyst is composed of calcium hydroxide, zinc oxide and zinc stearate. Radiopacity is provided by calcium tungstate, or in some cases by barium sulphate fillers. Calcium hydroxide should not be applied in a thickness greater than 0.5 mm, which would make it a liner.

Calcium hydroxide is considered to be bactericidal due to its high pH, approximately 12, which is provided by the catalyst. This alkaline property can cause cytotoxic effects to both the pulp and any bacteria in the preparation. Additionally, the acidic by-products of the bacteria are counteracted by the high pH. This high pH continues even after the material has set due, to hydroxyl ions that continue to leach out of the material when it comes in contact with the dentinal fluid. Calcium hydroxide can also irritate the pulp due to its high alkaline nature. This results in the formation of reparative dentine (a dentine bridge). This new dentine forms because CaOH can stimulate growth factors in the dentine matrix, and this process may occur more quickly when a resin-based calcium hydroxide formulation is used. Calcium as well as hydroxyl ions play an important role on the pulpal healing by modifying the environmental pH in the zone of inflammation to levels favourable for pulp matrix mineralization.

There are light-cured resin-based versions of calcium hydroxide and such formulations are not harmful to the pulp but do not show any antibacterial characteristics. They have a demand set and are less soluble than the self-cured products. The high solubility of conventional CaOH materials requires that clinicians ensure that restoration margins are sealed.

#### **Properties of Calcium Hydroxide**

- Low thermal conductivity.
- Stimulates the production of irregular secondary (tertiary) dentine pH of 11–12 (i.e. alkaline).
- Bactericidal properties.
- Highly soluble (not applicable to resin version).

#### **Advantages**

- Easily manipulated.
- Stimulates the formation of irregular secondary (tertiary) dentine.

#### **Disadvantages**

- Moisture sensitive.
- Low strength.

#### **Indications for use of Calcium Hydroxide**

- Protects the pulp from chemical irritation by its sealing ability.
- Stimulates the production of reparative or secondary dentin.
- Compatible with all types of restorative materials.
- Used in the deepest portion of cavity preparation.
- For use with direct or indirect pulp capping.
- Only used when within 1–2 mm of pulp or direct pulp capping.
- May be used underneath a base.

**Contraindications.** Cannot be applied thick enough to provide thermal protection for the pulp due to poor strength.

Operators will find that CaOH is easy to manipulate, hardens rapidly when applied in thin layers, provides a relatively good seal and has positive effects on both carious dentine and exposed pulp. Unfortunately, it is low in strength, undergoes plastic deformation and is highly soluble in water, and resins-based restorative materials will not bond to conventional CaOH.

Calcium hydroxide products are available in either a paste-paste version or a liquid formulation. Trade names: examples of calcium hydroxide paste-paste products are Dycal, VLC Dycal (Caulk Dentsply, Milford, DE, USA), Life (Kerr, Orange, CA, USA). Liquid versions of CaOH are the resin-based products Hydroxyliner (George Taub Products, Jersey City, NJ, USA) and Timeline (Caulk Dentsply, Milford, DE, USA).

Hydroxyliner and Hydroxyliner TC – Calcium hydroxide cavity liner (Taub Products, USA).



Figure 9. liners with Calcium hydroxide.

**Zinc oxide eugenol (ZOE).** The powder is composed of zinc oxide (70% by weight) with rosin added to reduce the brittleness of the set material. The eugenol is in the liquid portion, derived from oil of cloves (one of the «essential oils»). The eugenol is bactericidal on its own, but is more potent when combined with zinc oxide.

ZOE has been available for over 100 years. Despite having a pH of about 7 and having a sedative effect on the pulp, the eugenol can be toxic to the pulp, especially when present in high concentrations. It is for this reason that ZOE should not be placed in direct contact with the pulp.

Eugenol is released from the mixture by hydrolysis. The wet dentine causes enough eugenol to be released to form a concentration gradient that kills bacteria, but does not damage the pulp. Hume showed that the dentine protects the pulp from chemical irritation and as the remaining dentine thickness increases, so does the protection.

Even though ZOE does not bond to the tooth, it does afford an excellent marginal seal, which is better when a lower powder-liquid ratio is used. The advantage of this seal is the prevention of diet-derived substrate from reaching the micro-organisms found below the restoration. This results in the reduction of both acid production and of the formation of secondary



caries. Essentially, ZOE inhibits bacterial cell metabolism, the end result being a low incidence of postoperative sensitivity.

Hydrolysis of zinc oxide precedes a reaction between the resulting zinc hydroxide and eugenol, and this allows the ZOE mixture to set. The reaction occurs in the presence of water acting as a catalyst, which is why the reaction occurs faster when wet than when no moisture is present.

ZOE is not marketed as a cavity liner, but as a base (as well as other uses not relevant to this paper). Some products contain polymethylmethacrylate, which is incorporated in order to strengthen the material, making it more appropriate for use as a cavity base.

There are no ZOE products that are marketed for use as a liner. An example of ZOE as a base is IRM (Intermediate Restorative Material; Caulk Dentsply, York, PA, USA), available both in powder-liquid and encapsulated versions.

**Mineral trioxide aggregate (MTA)**, a material currently being used in pulp therapy, has been demonstrated to provide an enhanced seal over the vital pulp and is non-resorbable. MTA has been used experimentally for a number of years and was approved for human usage by the FDA in 1998.

MTA is an ash-colored powder made primarily of fine hydrophilic particles of tricalcium aluminate, tricalcium silicate, silicate oxide, and tricalcium oxide. When the material is hydrated it becomes a colloidal gel. The main components of MTA are calcium phosphate and calcium oxide. The material sets in approximately 3-4 hours, and, for radiopacity, vismuth oxide powder has been added, achieving a radiopacity similar to gutta percha. The initial pH of MTA when hydrated is 10.2 and the set pH is 12.5, which is comparable to that of calcium hydroxide. MTA has been found to have a set compressive strength of about 70 MPA. This is approximately equal to that of IRM but much less than amalgam (311 MPA).

MTA stimulated the release of cytokines and the production of interleukin, new bone formation.

The material has also been shown to have antimicrobial properties, have low cytotoxicity

The setting ability of MTA is uninhibited by blood or water. Place a 1-1.5 mm thick layer of freshly mixed MTA directly over the exposed pulp. Place a wet, thinned, flattened cotton pellet over the MTA.

Examples: ProRoot™ (Tulsa Dental), Grey MTA PlusR \_Avalon Biomed.

**Indications:**

- Pulp capping.
- Cavity lining.
- Pulpotomies.

### Question 6. Treatment of deep caries lesions.

**1) Traditional complete caries removal (removal of all soft and leathery dentin).** The traditional caries removal technique involves the removal of all soft and leathery dentin until hard dentin is reached before placing a final restoration. In shallow to moderate dentinal cavitated caries lesions (that radiographically appear to extend less than 75 percent into the dentin) this technique is often used without the risk of exposing the pulp.

Deep caries lesions are cavitated caries lesions that radiographically extend more than 70 to 75 percent into dentin. When the traditional caries removal technique is used to treat the deep caries lesions of vital asymptomatic teeth, the risk of pulp exposure is high. Complete removal of the soft and leathery dentin may cause a pulpal exposure, introducing bacteria into the pulp. Such outcomes require either root canal therapy or extraction. Evidence indicates that the traditional complete caries removal procedure may be detrimental to the pulpo-dentinal complex and does not take into consideration the biological natural response of the tooth to the caries stimulus.

**Table 34. Indications, advantages and limitations for complete caries removal**

<i>Indications</i>	<i>Advantages</i>	<i>Limitations</i>
Indicated for shallow to moderate caries lesions with no risk of pulp exposure	One visit (time for dentist and patient)	Invasive treatment for deep caries lesions
Only for deep caries lesions when:		Excessive removal of tooth structure
– Tooth is treatment plan for indirect restoration		Does not follow a biological approach to maintain pulp vitality
– Key Teeth abutment for FPD or RPD		
– Tooth will benefit from elective RCT		High risk of pulp exposure in deep caries lesions
– To clarify diagnosis when suspecting irreversible pulpitis		
– By patient informed decision		
FPD: Fixed Partial Denture RPD: Removable Partial Denture RCT: Root Canal Treatment		

For deep lesions, this lack of clinical evidence may result in an excavation that risks pulp exposure.

In a tooth with a deep caries lesion, no history of spontaneous pain, normal responses to thermal stimuli, and a vital pulp, a deliberate, incomplete

caries excavation may be indicated. This procedure is termed indirect pulp capping (also referred to as stepwise caries excavation or partial caries excavation) and is supported by a large body of evidence. In brief, indirect pulp capping consists of complete caries excavation peripherally to a sound, caries-free DEJ; axially and pulpally, caries is excavated to within approximately 1 mm of the pulp; a glass ionomer (e.g., Fuji IX, GC, Alsip, IL) sedative restoration or a definitive restoration is then placed. The glass ionomer is used when the clinician anticipates a follow-up appointment will be needed to re-enter the preparation and complete the caries excavation.

Pulp exposures can be due to mechanical reasons, caries or trauma. When the pulp exposure of a vital asymptomatic tooth is mechanical or due to trauma, the Direct Pulp Capping procedure has been used in an attempt to preserve tooth vitality. After rinsing and disinfecting the exposure site, a liner usually from calcium hydroxide or MTA material is placed directly over the exposed pulp followed by a sealing liner of resin modified glass ionomer and the final restoration.

It is thought that pulp exposures due to trauma or mechanical reasons (iatrogenic) have a better success rate than a caries exposure since there is no bacterial contamination. When the pulp is exposed due to caries, the bacterial contamination will cause inflammation, decreasing the healing ability of the pulp and resulting in irreversible damage or necrosis. In deep caries lesions of asymptomatic, vital restorable teeth the pulp exposure should be avoided; it is preferable to use an incomplete caries removal approach rather than the direct pulp treatment.

The use of incomplete caries removal techniques have been proposed based on the deeper understanding of the biological response of the tooth to caries stimulus and the structural changes that occur as a protective response of the tooth to bacterial invasion.

The incomplete caries removal technique involves the partial removal of soft caries infected dentin starting peripherally (at the DEJ) and the sealing of the remaining caries lesion with an interim or final restoration (in one or two visits) with the goal to seal the partially demineralized affected dentin and arrest or reverse caries lesion progression.

**2) *The Conservative Approach to Partial Caries Removal.*** The most widely known and used techniques are the Indirect Pulp Treatment (formerly termed as "capping") and Stepwise Caries removal. They differ in the amount of soft dentinal tissue removed, number of appointments involved (one or two), and restorative materials. Each technique has indications, advantages and limitations. To properly utilize these techniques for different clinical situations, tooth and pulpal diagnosis is crucial, as well as the understanding of the caries lesion activity, and related changes in the dental structures.

### **Stepwisecaries removal**

Stepwise excavation is an alternative technique for removal of deep caries lesions that radiographically involve 75 percent or more of the total dentin thickness and do not already penetrate to the pulp. The purpose of the stepwise excavation approach is to change the cariogenic environment of deep caries lesions by removing only the soft wet infected dentin and then sealing the remaining demineralized dentin with an interim restoration. The goal is to arrest the active caries lesion and stimulate dentinal tubule sclerosis and the formation of reparative dentin while maintaining pulp vitality.

**Table 35. Indications, advantages and limitation of stepwise treatment**

<i><b>Indications</b></i>	<i><b>Advantages</b></i>	<i><b>Limitations</b></i>
Deep caries lesions in vital asymptomatic (reversible pulpitis & non periradicular pathosis), restorable teeth	Preserves tooth structure	Time (2 visits)
Active\soft\rapid progressive lesions	Promote remineralization of soft remanent dentin	Cost (patient may need to pay for provisional and final restorations)
Usually in compliant young patients	Thicker remaining dentin over pulpal and axial walls, less likely to cause pulp exposure	May not be covered by third party payers
Patient OK with 2 steps and 6.0 months re-entry	Biological approach promoting tubule sclerosis and formation of reparative, reactionary dentin	Patient compliance (patient may not come back for final restorations)
Primary lesions or RCA in shallow restorations with thick RDT	Provides time to clarify pulpal diagnosis	Risk of pulp exposure at re-entry
In caries control cases (multiple teeth with rampant caries)		When compromise future restorability. Key teeth, abutments and teeth in need of large indirect restorations
RCA: Recurrent Caries RDT: Remaining Dentinal Thickness		

### **Technique for stepwise excavation**

After a detailed evaluation and correct case selection using the previous criteria, the stepwise approach is performed in two separate appointments with an interval of six to eight months.

#### **First Appointment**

1. Inform the patient about the treatment options including benefits and possible drawbacks. Allow the patient to be part of the decision.
2. Rubber dam isolation is highly recommended.

3. Access to the caries lesion, peripheral excavation should be completed by cleaning the DEJ, removing the very soft, necrotic and infected dentin and leaving the soft, discolored yellow or dark leathery dentin over the pulpal floor and axial walls. Avoid excavating close to the pulp during this first step to reduce the risk of pulp exposure.
4. Restore with a temporary glass ionomer material. For example, first use Fuji Triage GC® as a liner (color coded for reentry) and then place Fuji IX or Fuji II LC GC® as a restorative material.
5. Schedule appointment (six to eight months) for re-entry.

Second Appointment (Re- Entry)

1. Re-evaluate history of symptoms.
2. Clinical exam to evaluate for swelling or tenderness.
3. New periapical radiograph to verify lack of pathosis.
4. Pulp vitality tests.
5. If all of the above are normal isolate teeth, preferable with Rubber Dam.
6. Remove the sedative filling peripherally first and then be especially careful when approaching the Fuji Triage liner. The Fuji Triage does not need to be completely removed if arrested and well-sealed dentin is observed, it may be maintained over pulpal and axial wall to prevent pulp exposure.
7. Dentin assessment (peripheral) and careful removal of any remaining soft dentin.
8. Placement of glass ionomer liner over the exposed dentin (Vitrebond 3M®)
9. Restore with the material of choice for final restoration.
10. Six month recall for evaluation of vitality tests and periapical radiograph.
11. Continue with similar annual recalls.

The difference between Stepwise Caries Removal and Indirect Pulp Treatment is that the stepwise procedure is performed in two visits (usually months apart). In the first visit, the soft necrotic carious dentin is removed partially and peripherally and the tooth is sealed with an interim restoration. The time interval between the two visits allows remineralization to occur and tertiary dentin to develop. At the second visit, the tooth is re-entered, the residual affected soft dentin is removed and the final restoration is placed. Two recent systematic reviews, Rickets et al. and Schewendicke et al. have compiled and analyzed the evidence suggesting that there are potential benefits to reducing the risk of pulp exposure in using either one or two steps techniques compared with complete caries removal. There is still a need of more evidence and good standardize clinical research to determine whether is necessary to re-enter.

### Indirect pulp treatment (IPT)

ITP consists in the removal of all peripheral soft dentin of the deep caries lesion, leaving a thin residual layer (0.5mm–1.0mm) of leathery affected dentin over the pulpal floor or axial wall followed by a liner and placement of the final restoration with the goal of preventing pulp exposure.

Use of incomplete caries removal techniques significantly decreases the risk of pulp exposure in deep caries lesions compared with the traditional complete caries removal procedure, and these restorations have shown similar success.

If there is any remaining bacteria after the caries is partially removed, the placement of a restoration providing a good seal will arrest the lesion progression by isolating the bacteria from the substrate and decreasing acid production.

Table 36. **Indications, advantages and limitation of indirect pulp treatment**

<i>Indications</i>	<i>Advantages</i>	<i>Limitations</i>
Deep caries lesions in vital asymptomatic (reversible pulpitis & non periradicular pathosis), restorable teeth.	One visit (time for dentist and patient)	More structure removed than Stepwise technique but less than complete caries removal technique
Presence of sclerotic dentin in a chronic slow progressing lesion	Cost	Greater risk of pulp exposure by accident than SWE (1st appointment)
Desire for final restoration in same appointment	Insurance coverage	Less RDT is left (around 0.5 mm of residual affected dentin)
RCA under deep existing restoration with thin RDT		
Asymptomatic patient that deny RCT and have above indications		No time interval to clarify diagnosis or promote tubule sclerosis
By patient informed decision		
RCA: Recurrent Caries RDT: Remaining Dentinal Thickness RCT: Root Canal Treatment		

### Technique steps for indirect pulp treatment

1. No history of spontaneous pain.
2. Proper Diagnosis: EPT (Electric Pulp Test).
3. Periapical radiograph with normal periapical structures
4. Good Isolation (Preferable Rubber Dam).
5. Peripheral caries at the DEJ removed while maintaining thin residual caries dentin over pulpal and axial walls.
6. Clean DEJ at cavosurface margin to achieve a good restoration seal.

7. Finish cavity preparation (clean and smooth walls) with design depending on material selection.
8. Liner placement (either calcium hydroxide, resin modified glass ionomer (ex: Vitrebond™ 3M) or a resin-modified calcium silicate filled liner® Theracal (Bisco).
9. Final restoration providing good seal.
10. Follow-up\Recall within three to six months.

### **Tests to the topic**

#### **1. Name the main principles of tooth preparation**

- a. To restore function.
- b. To prevent further spread of an active lesion which is not amenable to preventive measures.
- c. To preserve pulp vitality.
- d. Provide access to pulpal chamber.

#### **2. What is the approach of cavity formation, that has main principle «extension for prevention»?**

- a. Black's technique.
- b. Technique of adhesive preparation.

#### **3. What are types of dental bors?**

- a. Diamond.
- b. Steel.
- c. Carbide.
- d. Hard-allow.
- e. All of the above.

#### **4. What parts does any rotary cutting instrument include?**

- a. Shank.
- b. Neck.
- c. Head.
- d. All of the above.

#### **5. What are the stages of carious cavity preparation?**

- a. Opening of cavity.
- b. Enlargement of cavity.
- c. Necrectomy
- d. Formation the cavity
- e. All of the above.

**6. Causes of pulp damage during tooth and treatment are**

- a. Heat generated by rotary instruments.
- b. Some ingredients of various materials.
- c. Thermal changes conducted through restorative materials.
- d. The ingress of different products and bacteria through microleakage.
- e. All of the above

**7. What are functions of Liners?**

- a. Barrier that protects dentin from irritants agents from either the restorative materials or oral fluids.
- b. Some thermal protection.
- c. To restore form and function of a tooth.

**8. Name properties of CaOH materials**

- a. Stimulates the production of irregular secondary (tertiary) dentine.
- b. PH of 11–12 (i.e. alkaline).
- c. Bactericidal properties.
- d. Insoluble.

**9. What are disadvantages of CaOH materials?**

- a. Moisture sensitive.
- b. pH of 11–12 (i.e. alkaline).
- c. Low strength.
- d. Biocompatibility.

**10. What are introductions to use of MTA materials?**

- a. Pulp capping.
- b. Cavity lining.
- c. Restoration of tooth crown.
- d. Treatment of hypersensitivity of dentin.



## **LESSON 12. DIAGNOSIS AND TREATMENT OF OCCLUSION SURFACES CARIES. SELECTION OF FILLING MATERIAL**

The questions to be studied for the learning of the topic:

1. Lesions of Black's Class I. Location. Clinical characteristics.
2. Diagnosis of carious lesions of Class I.
3. Peculiarities of Black's preparation of carious lesions of Class I.
4. Class I Amalgam Restoration.
5. Class I Composite Restoration.

### **Question 1. Lesions of Black's Class I. Location. Clinical characteristics.**

Locations of lesions of Class I include:

- Occlusal surface of molars and premolars
- Lingual surface of anterior teeth
- Occlusal two thirds of buccal and lingual surfaces of molars and premolars, i.e. blind pits of teeth.

The occlusal surface is characterized by the pit and fissure systems, a favorable biofilm stagnation area where the bacterial accumulations receive the best protection against functional/mechanical wear (mastication, attrition, abrasion from brushing, flossing or toothpicks). Those aspects contribute to the high prevalence of caries on occlusal surfaces both in the primary and permanent dentition.

The complex anatomy of the occlusal surfaces requires professional special attention and deep understanding of how lesions develop on this surface. It is known that the deepest part of the fissure usually harbors non-vital bacteria or calculus. An enamel caries lesion begins along the pits and fissures through acids diffusion from bacterial metabolism in the biofilm. This diffusion occurs through the side walls of the pits and fissures, guided by prisms direction and striae of Retzius. Histologically, the lesion forms in three dimensions and assumes the shape of a cone, with its base toward the enamel-dentin junction. Acids lead to the demineralization underneath the enamel surface and there is an enlargement in intercrystalline spaces, increasing its permeability. Over time, the surface porosity has increased and leads to a considerable increase of the lesion body (a subsurface lesion starts to form). Occlusal enamel breakdown is the result of further demineralization, thus leading to cavity formation.

**Question 2. Diagnosis of carious lesions of Class I**  
**Main and additional methods of diagnosis of carious lesions of**  
**Class I**

**The main methods of diagnosis**

**1. Questioning:**

- ✓ Complaints.
- ✓ Patient history.
- ✓ Medical history.

**2. Clinical Examination:**

Probing.

- ✓ To determine of roughness of enamel surface.
- ✓ To determine of dentin density (soft or hard) at a bottom and a walls of caries cavity.
- ✓ To determine of sensitivity (a bottom and a walls of caries cavity).

Drying. Reaction on thermal agents (cold, heat).

For the purpose of caries management, individual tooth surfaces are categorized and described, based on an evaluation of each surface affected. For pits and fissures, the evaluation criteria are as follows.

**Table 37. Pits and fissures evaluation criteria**

<b>Tooth surface</b>	<b>Evaluation criteria</b>	<b>ICDAS code</b>
Sound surfaces	No visible caries when viewed clean and dry. Non-carious white or brown marks on tooth surfaces must be differentiated from early caries lesions.	0
Initial stage caries	Characterized by the first visual change in enamel (seen only after prolonged air drying or restricted to the confines of a pit or fissure). <b>OR</b> A distinct visual change in enamel (seen on a wet or dry surface).	1  2
Moderate stage caries	Characterized visually by either localized enamel breakdown (without visual signs of dentinal exposure). - Enamel breakdown is often viewed best when the tooth is air dried. <b>OR</b> An underlying dark shadow from dentin. - Shadowing from dentinal caries is often	3  4

	best seen with the tooth surface wet.	
Extensive stage caries	Characterized by distinct cavitation exposing visible dentine. - Lesions exhibiting cavitation Involving less than half the tooth surface - Lesions involving half of the tooth surface or more	5  6

### **The additional methods of diagnosis of carious lesions of Class I**

- Radiologic and clinical examination
- Emerging diagnostic techniques
- Fluorescence
- Fiber-optic transillumination (FOTI)
- Digitally imaging fiber-optic transillumination (DIFOTI)
- Electrical conductivity.

***Bite-wing (BW)*** films for proximal decay detection.

***Directions in caries diagnosis for occlusal caries.*** Caries researchers recommend against using a sharp explorer when examining pits and fissures. Histological evidence shows the explorer can disrupt incipient caries and bacteria can be moved from groove to groove by a sharp explorer. Reliance on a sharp explorer has the potential to underutilize other detection methods and leads to a false positive diagnosis of occlusal caries.

Visualization of pit and groove areas for discoloration under the enamel is essential for pit and fissure diagnosis. Teeth must be clean, dry, and well illuminated to be properly evaluated. With the greater exposure to fluorides and more frequent placement of sealants deep caries can exist without a visible indication. Radiographs, in the past considered of limited use in the detection of occlusal caries, have been shown to be valuable in detecting deep dentin caries beneath pit and fissures and sealants.

***Bitewing radiography diagnosis*** is that it is not invasive, and does not damage tooth structure like an incorrectly used dental probe might. Radiographs can also be filed and reexamined at a later date to compare with a more recent image to detect whether a lesion is progressing or not.

***Limitations of bitewing radiography diagnosis.*** Besides concerns about low-dose radiation and variations in how images are interpreted by dentists, the main limitation is that the validity in diagnosing early lesions is rather low. Also, the bitewing radiograph cannot always distinguish between sound surfaces, those with initial caries activity and cavitated lesions, or non-carious demineralizations, so clinical inspection is still needed to determine what is happening to the tooth. Bitewing radiographs also tend to underestimate the depths of lesions, so a lesion that appears confined to the

inner enamel on an image is often actually in the dentin, and this can lead to insufficient or improper treatment.

However, digital radiography is replacing radiography based on film. It has been proven as accurate as traditional radiography for detecting caries, and it comes with additional advantages of using a lower radiation dose, being less time-consuming, and does not require wet chemicals in the processing of the image.

**Digital radiography** – which is increasingly replacing bitewing radiography and that is as accurate as film for the detection of caries lesions.

**Digital image enhancement** – which studies show can provide superior results to radiographs when enhanced correctly but takes a significant amount of technical skill.

### **Question 3. Peculiarities of Black's preparation of carious lesions of Class I.**

According to Black's principles of preparation, a cavity of the I class should be: with straight walls at right angle to the bottom, a form of the cavity should be cylindrical, box-shaped, rhombic, X-like.

Regardless of cavity location, there are common stages of dental hard tissues preparation, which are come to:

**Disclosure** (opening and enlargement) of cavity (is conducted by using round-shaped, fissures burs, burs that is chosen, should have the size of the working end not bigger than the entrance aperture of this cavity).

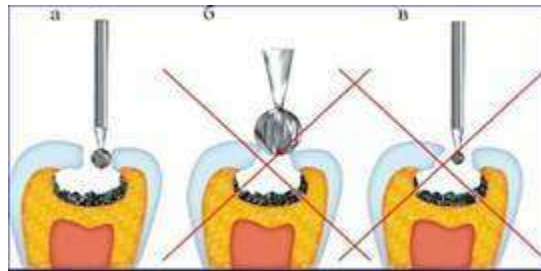
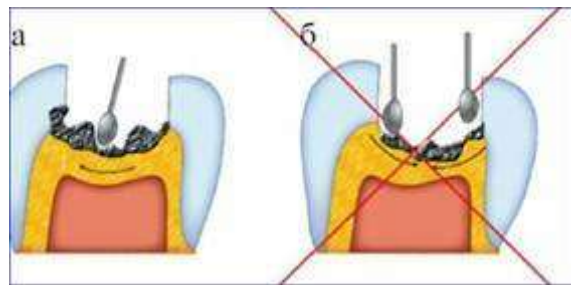


Figure 10. Correct and incorrect opening of cavity.

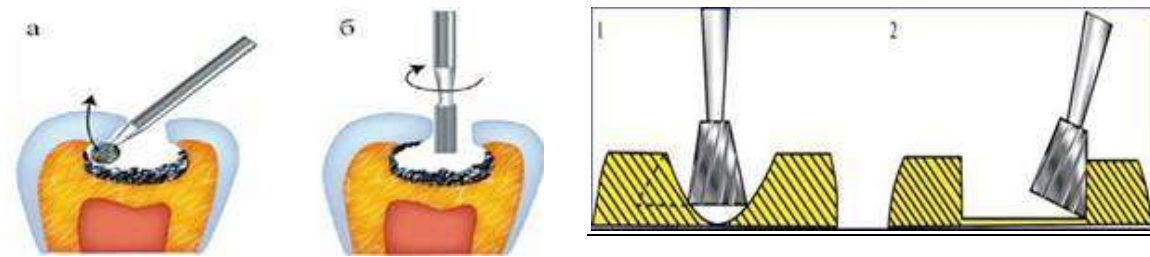
**Necrectomy** is a removal of the necrotic dental hard tissues from carious cavity. There are total and partial necrectomy. Total -is complete removal of necrotic dentin from the walls and bottom of the cavity. Partial - is complete removal of necrotic dentin from walls and partly from the bottom of the cavity. Partial necrectomy is allowed in the case of deep dental caries, when the bottom of the cavity is very thin and there is a danger of the pulp horn disclosure. In this case is permitted to leave on the bottom of the cavity a dense pigmented dentin, and in the course of acute deep caries – is allowed to leave a small layer of softened dentin with the next remineralization

influence on it. Necrectomy is done by the round-shaped burs and the excavator.

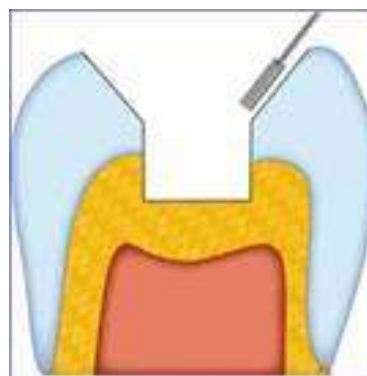


*Figure 11. Correct and incorrect directions of bor movement during of necrotomy.*

Formation the cavity for fillings (is done with fissures, inverted-cone and cone-shaped burs).



*Figure 12. Directions of movement of different bors during offormation the cavity.*

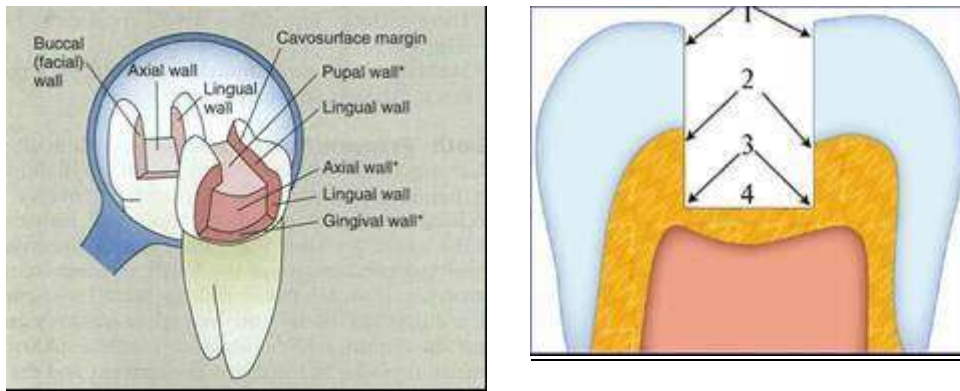


*Figure 13. Smoothing the edges of enamel.*

**Cavity wall: Side or surface of a tooth prepared for restoration.**

- Internal wall: Cavity wall that does not extend to the external tooth surface.
- External wall: Portion of the tooth preparation that extends to the external tooth surface, named according to the tooth surface involved: distal, mesial, facial, lingual, and gingival.

- Axial wall: Internal wall of prepared tooth that runs parallel to the long axis of the tooth.
- Pulpal wall: Internal wall of prepared tooth that is perpendicular to the long axis of the tooth; also known as the pulpal floor.
- Line angle: Angle formed by the junction of two walls in a cavity preparation.



*Figure 14. Cavity wall.*

There are general rules for the preparation of cavities according to Black's principles of preparation:

1. Transition the bottom of the cavity (the surface which is turned to a pulp) to the side wall should be at right angle.
2. Transition of one wall to another should be at an angle –the form of the cavity-box- shaped form (except the V class).
3. Enamel edges should be straight and smooth.
4. Bottom of the cavity should be flat or somewhat reminding the form of the occlusal surface of the tooth.

Dissection of tooth tissues for filling with composites materials is slightly different from the traditional preparation by Black. This is because the traditional preparation is used for mechanical fixing of fillings in the carious cavity. Composite materials have the ability to chemically bind to tissues, so there is no need to prepare a wall at right angles.

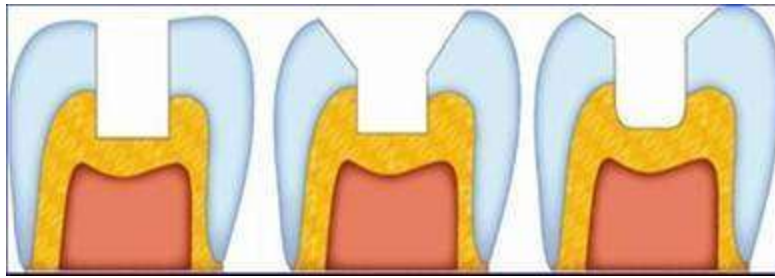


Figure 15. Variants of traditional preparation by Black and preparation for Composite materials.

#### Question 4. Class I Amalgam Restoration.

##### *Types of Amalgam Restorative Materials*

**Low-copper amalgam.** Low-copper amalgams were prominent before the early 1960s. When the setting reaction occurred, the material was subject to corrosion because a tin-mercury phase (gamma-two) formed. This corrosion led to the rapid breakdown of amalgam restorations. Subsequent research for improving amalgam led to the development of high-copper amalgam materials.

**High-copper amalgam.** High-copper amalgams are the materials predominantly used today in the World. The increase in copper content to 12% or greater designates an amalgam as a high-copper type. The advantage of the added copper is that it preferentially reacts with the tin and reduces the formation of the more corrosive phase (gamma-two) within the amalgam mass. This change in composition reduces possible deleterious corrosion effects on the restoration. However, enough corrosion occurs at the amalgam-tooth interface to result in the successful sealing of the restoration. These materials can provide satisfactory performance for more than 12 years. High-copper materials can be either spherical or admixed in the composition.

**Spherical amalgam.** A spherical amalgam contains small, round alloy particles that are mixed with mercury to form the mass that is placed into the tooth preparation. Because of the shape of the particles, the material is condensed into the tooth preparation with little condensation pressure. This advantage is combined with its high early strength to provide a material that is well suited for very large amalgam restorations such as complex amalgams or foundations.

**Admixed amalgam.** An admixed amalgam contains irregularly shaped and sized alloy particles, sometimes combined with spherical shapes, which are mixed to form the mass that is placed into the tooth preparation. The irregular shape of many of the particles makes a mass that requires more condensation pressure (which many dentists prefer) and permits this heavier condensation pressure to assist in displacing matrix bands to generate proximal contacts more easily.

***New amalgam alloys.*** Because of the concern about mercury toxicity, many new compositions of amalgam are being promoted as mercury free or low-mercury amalgam restorative materials. All with gallium or indium or alloys using cold-welding techniques are presented as alternatives to mercury-contain amalgams. None of these new alloys shows sufficient promise to become a universal replacement for current amalgams materials.

***Important properties.*** The linear coefficient of the thermal expansion of amalgam is 2,5 times greater than that of tooth structure, but it closer than the linear coefficient of thermal expansion composite. Although the compressive strength of high-copper amalgam is similar to tooth structure, the tensile strength is lower, making amalgam restorations prone fracture.

Usually, high-copper amalgam fracture is a bulk fracture, not a marginal fracture. All amalgams are brittle and have low edge strength. The amalgam material must have sufficient bulk (usually 1 to 2 mm, depending on the position within the tooth) and a 90-degree or greater marginal configuration.

Creep and flow relate to the deformation of a material under load over time. High-copper amalgams exhibit no clinically relevant creep or flow. Because amalgam is metallic in structure, it also is a good thermal conductor. An amalgam restoration should not be placed close to the pulpal tissues of the tooth without the use of a liner or base (or both) between the pulp and the amalgam.

***Amalgam restorations.*** Amalgam functions as a direct restorative material because of its easy insertion into a tooth preparation and, when hardened, its ability to restore the tooth to proper form and function. The tooth preparation form not only must remove the fault in the tooth and remove weakened tooth structure, but it must also be formed to allow the amalgam material to function properly.

There required tooth preparation form must allow the amalgam to:

- 1) possess a uniform specified minimum thickness for strength;
- 2) produce a 90-amalgam angle (butt-joint form) at the margin;
- 3) be mechanically retained in the tooth.

Amalgam is less technique sensitive or operator sensitive compared with composite. Amalgam is used for the restoration of many carious or fractured posterior teeth and in the replacement of failed restorations.

Material qualities and properties for Class I amalgam restorations include the following:

- Strength
- Longevity
- Ease of use
- Clinically proven success



**Advantages.** Primary advantages are the ease of use and the simplicity of the procedure. The placing and contouring of amalgam restorations are generally easier than those for composite restorations.

**Disadvantages.** The primary disadvantages of using amalgam for Class I defects are:

1) Amalgam use requires more complex and larger tooth preparations than composite resin,

2) Amalgams may be considered to have a non-esthetic appearance by some patients.

*Clinical indications for direct amalgam restoration* – moderate to large restoration, especially restoration that involve heavy occlusion, can not be isolated well, or extend onto the root surface.

*Contraindications for amalgam restoration* – amalgams contraindicated in patients who are allergic to the alloy components. The use of amalgam in more prominent esthetic areas of the mouth is usually avoided.

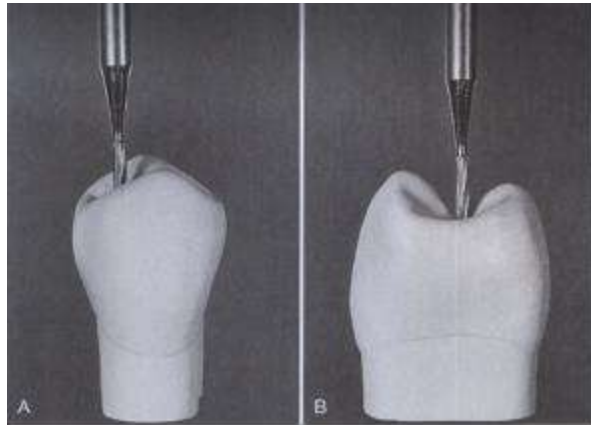
### **Clinical technique for class I amalgam restorations**

***Conservative class I amalgam restorations.*** Conservative tooth preparation is recommended to protect the pulp, preserve the strength of the tooth, and reduce deterioration of the amalgam restoration. Such conservative preparation saves the tooth structure, minimizing pulpal irritation and leaving the remaining tooth crown as strong as possible. Conservative preparation also enhances marginal integrity and restoration longevity.

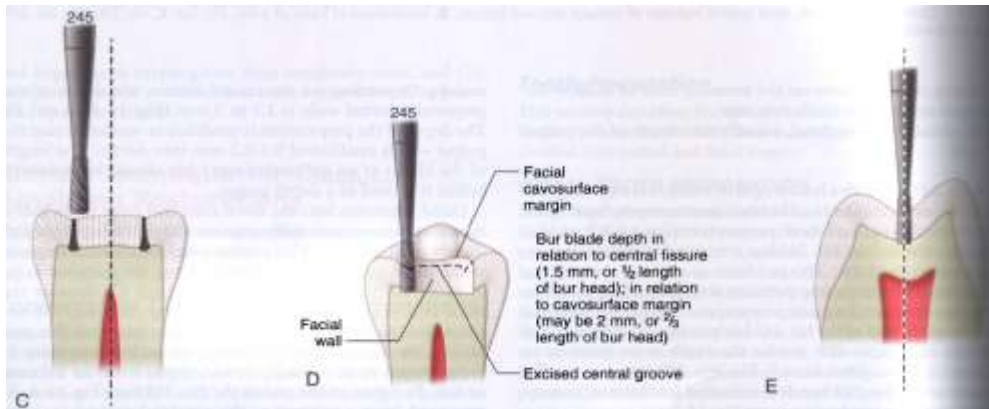
***Initial clinical procedures.*** After the onset of profound anesthesia, isolation with the rubber dam is recommended to gain control over the operating field and for mercury hygiene. For a single maxillary tooth, where caries is not extensive, adequate control of the operating field may be achieved with cotton rolls and high-volume evacuation. A pre-operative assessment of the occlusal relationship of the involved and adjacent teeth also is necessary.

***Initial tooth preparation*** is defined as establishing the outline form by extension of the external walls to sound tooth structure while maintaining a specified, limited depth (usually just inside the dentinoenamel junction [DEJ] and providing resistance and retention forms. The outline form for the Class I occlusal amalgam tooth preparation should include only the defective occlusal pits and fissures (in a way that sharp angles in the marginal outline are avoided). The ideal outline for a conservative amalgam restoration incorporates the following resistance form principles that are basic to all amalgam tooth preparations of occlusal surfaces. These principles allow margins to be positioned in areas that are sound and subject to minimal forces while conserving structure to maintain the strength and health of the tooth. The resistance principles are as follows:

- Extending around the cusps to conserve tooth structure and prevent the internal line angles from approaching the pulp horns too closely.
- Keeping the facial and lingual margin extensions as minimal as possible between the central groove and the cusp tips.
- Extending the outline to include fissures, placing margins on relatively smooth, sound tooth structure.
- Minimally extending into the marginal ridges (only enough to include the defect) without removing dentinal support.
- Eliminating a weak wall of enamel by joining two outlines that come close together (i.e.,  $<0.5$  mm apart).
- Extending the outline form to include enamel undermined by caries.
- Using enameloplasty on the terminal ends of shallow fissures to conserve tooth structure
- Establishing an optimal, conservative depth of the pulpal wall



*Figure 16. Directions of instrument.*



*Figure 17. Stages of preparation:*

*A, No. 245 bur oriented parallel to long axis of tooth crown for entry as viewed from lingual aspect.*

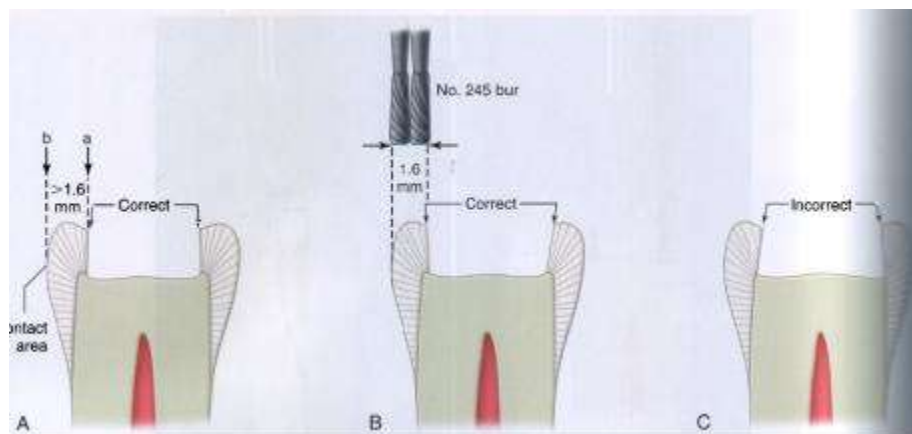
*B, The bur positioned for entry as viewed from the distal aspect.*

*C, The bur is positioned over the most carious pit (distal) for entry. The distal aspect of the bur is positioned over the distal pit.*

*D, Mesiodistal longitudinal section. Relationship of head of No. 245 bur to excised central fissure and cavosurface margin at ideal pulpal floor depth, which is just inside the dentinoenamel junction (DEJ).*

*E, Faciolingual longitudinal section. Dotted line indicates the long axis of tooth crown and the direction of the bur.*

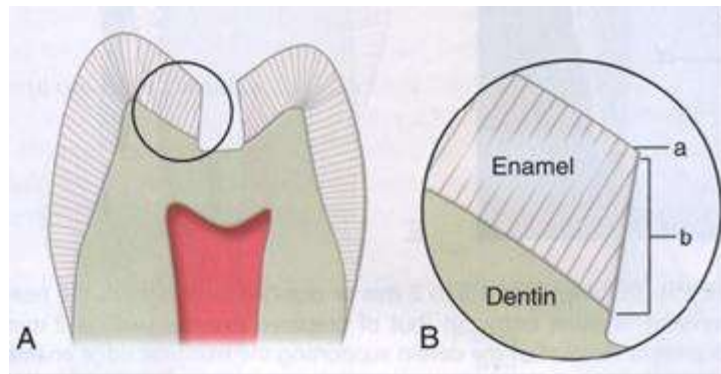
The direction of the mesial and distal walls is influenced by the remaining thickness of the marginal ridge as measured from mesial or distal margin to the proximal surface.



*Figure 18: A. Mesial and distal walls should converge when the distance from a to b greater than 1,6 mm.*

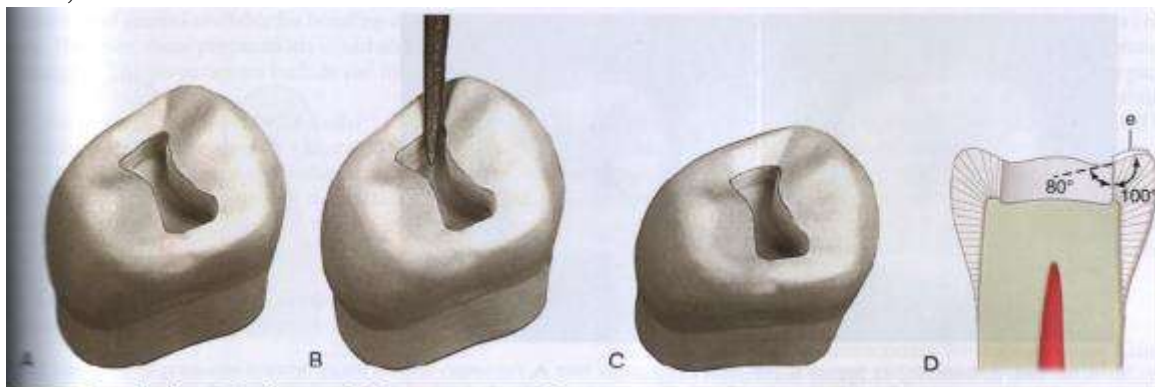
*B. When the operator judges that the extension will leave only 1,6 mm thickness (two diameters of N 245 bur) of marginal ridge (i. e., premolars) as illustrated, the mesial and distal walls must diverge occlusally to conserve ridge-supporting dentin.*

*C. Extending the mesial or distal walls to a two-diameter limit diverging the wall occlusally undermines the marginal ridge enamel.*



*Figure 19. A and B, The ideal and strongest enamel margin is formed by full-length enamel rods (a) resting on sound dentin supported on the preparation side by shorter rods, also resting on sound dentin (b).*

The ideal and strongest enamel margin is formed by full-length enamel rods resting on sound dentin supported on the preparation side by shorter rods, also on sound dentin.



*Figure 20. Enameloplasty. A. Development defect at terminal end of fissure.*

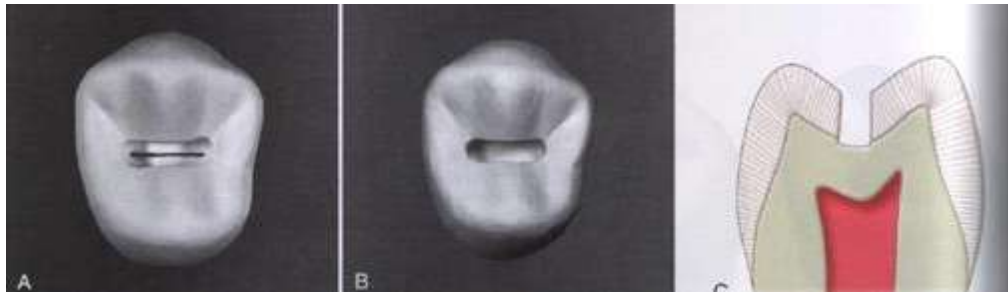
*B. Fine – grit diamond bor in position to remove the defect.*

*C. Smooth surface after enameloplasty.*

*D. The cavosurface angle should not exceed 100 degrees, and the margin-amalgam angle should not be less than 80 degrees.*



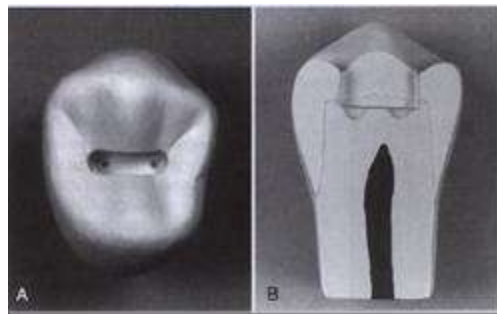
*Figure 21. Mesial fissure that cannot be eliminated by enameloplasty may be included in the preparation if the margins can be lingual of contact.*



*Figure 22. Removal of enamel fissure extending over most of the pulpal floor.*

*A, Full-length occlusal fissure remnant remaining on the pulp after the initial tooth preparation.*

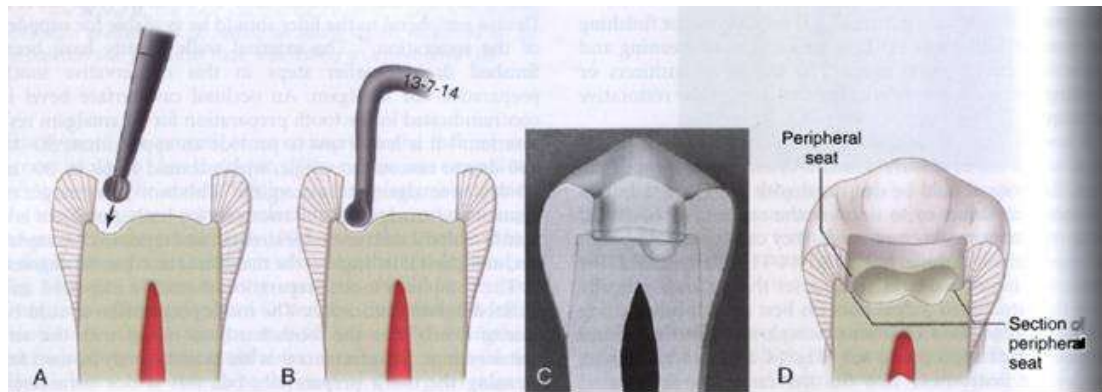
*B and C. The pulpal floor is deepened to a maximum depth of 2 mm to eliminate the fissure or uncover dentinal caries.*



*Figure 23. Removal of enamel pit and fissure and infected dentin that is limited to a few small pit-and-fissure remnants.*

*A, Two pit remnants on the pulpal floor after the initial tooth preparation.*

*B, Defective enamel and infected dentin have been removed.*



*Figure 24. A and B, Removal of dentinal caries is accomplished with round burs (A) or spoon excavators (B).*

*C and D, The resistance form improved with a flat floor peripheral to the excavated area or areas.*

The final tooth preparation includes:

- 1) Removal of remaining defective enamel and infected dentin on the pulpal floor,
- 2) Pulp protection, where indicated,

- 3) Procedures for finishing the external walls,
- 4) Final procedures of cleaning and inspecting the prepared tooth.

The removal of carious dentin should not affect the resistance form further because the periphery would not need further extension. In addition, it should not affect the resistance form if the restoration is to rest on the pulpal wall peripheral to the excavated area or areas. The peripheral pulpal floor should be at the previously described initial pulpal floor depth just inside the DEJ.

If the tooth preparation is of ideal or shallow depth, no liner or base is indicated. In deeper caries excavations (where the remaining dentin thickness is judged to be 0.5 to 1 mm), a thin layer (i.e., 0.5-0.75 mm) of a light-activated, resin-modified glass ionomer (RMGI) material should be placed. The RMGI insulates the pulp from thermal changes, bonds to dentin, releases fluoride, is strong enough to resist the forces of condensation, and reduces microleakage.

Dentin peripheral to the liner should be available for support of the restoration. The external walls already have been finished during earlier steps in this conservative tooth preparation for amalgam. An occlusal cavosurface bevel is contraindicated in the tooth preparation for an amalgam restoration. It is important to provide an approximate 90- to 100-degree cavosurface angle, which should result in 80- to 90-degree amalgam at the margins. This butt-joint margin of enamel and amalgam is the strongest for both. Amalgam is a brittle material with low edge strength and tends to chip under occlusal stress if its angle at the margins is less than 80 degrees. The completed tooth preparation should be inspected and cleaned before restoration. The tooth preparation should be free of debris after the tooth has been rinsed with the airwater syringe. Disinfectants that are available may be used for cleaning the tooth preparation, but this is not considered essential. A cotton pellet or a commercially available applicator tip moistened only with water is generally used.

#### ***Restorative Technique for Class I Amalgam Preparations***

The use of desensitizers or bonding systems is considered the first step of the restorative technique.

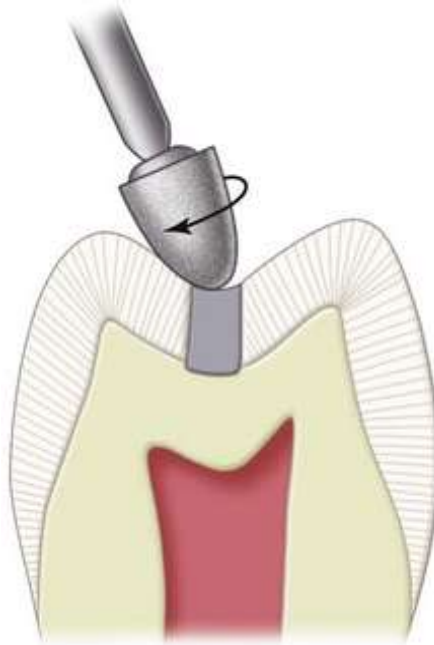
A dentin desensitizer is placed in the preparation before amalgam condensation. The dentin desensitizer is applied onto the prepared tooth surface according to manufacturer's recommendations; excess moisture is removed without desiccating the dentin; and then the amalgam is condensed into place. The dentin desensitizer precipitates protein and forms lamellar plugs in the dentinal tubules.

#### ***Insertion and carving of the amalgam***

The principal objectives during the insertion of amalgam are to condense the amalgam to adapt it to the preparation walls and the matrix



(when used) and produce a restoration free of voids. Thorough condensation helps to reduce marginal leakage. Optimal condensation also is necessary to minimize the mercury content in the restoration to decrease corrosion and to enhance strength and marginal integrity. Condensation of amalgam that contains spherical particles requires larger condensers than are commonly used for admixed amalgam. Smaller condensers tend to penetrate a mass of spherical amalgam, resulting in less effective force to compact or adapt the amalgam within the preparation. In contrast, smaller condensers are indicated for the initial increments of admixed amalgam because it is more resistant to condensation pressure. Because the area of a circular condenser face increases by the square of the diameter, doubling the diameter requires four times more force for the same pressure on a unit area.



*Figure 25. Polishing the amalgam. The stone's long axis or the bur's long axis is held at a right angle to the margin*

Carving should be accomplished so that opposing cusps contact on a surface that is perpendicular to the occlusal forces in maximum intercuspation. Occlusal contacts located on a cuspal incline or ridge slope are undesirable because they cause a deflective force on the tooth and should be adjusted until the resulting contact is stable (i.e., the force vector of the occlusal contacts should parallel the long axis of the tooth).

The patient has been instructed to close vertically in to maximum intercuspation. After placing the articulating paper over the tooth, the patient is asked to occlude lightly and to slide the teeth lightly from side to side. Any additional occlusal marks are evaluated, and undesirable contact areas are eliminated. Appropriate caution is indicated, as amalgam restorations carved

out of occlusion may result in undesirable tooth movement. Finally, the patient should be cautioned to protect the restoration from any heavy biting pressure for 24 hours.

Most amalgams do not require further finishing and polishing. These procedures are occasionally necessary, however, to:

- 1) Complete the carving;
- 2) Refine the anatomy, contours and marginal integrity;
- 3) Enhance the surface texture of the restoration;

Additional finishing and polishing procedures for amalgam restorations are not attempted within 24 hours of insertion because crystallization is incomplete.

If used, these procedures are often delayed until all of the patient's amalgam restorations have been placed, rather than finishing and polishing periodically during the course of treatment. An amalgam restoration is less prone to tarnish and corrosion if a smooth, homogeneous surface is achieved.

Polishing of high-copper amalgams is less important than it is for low-copper amalgams because high-copper amalgams are less susceptible to tarnishing and marginal breakdown.

### **Question 5. Class I Composite Restoration.**

#### **Advantages**

The advantages of composite as a Class I direct restorative material relative to other restorative materials are:

1. Esthetics.
2. Conservative tooth structure removal.
3. Easier, less complex tooth preparation.
4. Insulation.
5. Decreased microleakage.
6. Increased short-term strength of remaining tooth structure.

#### **Disadvantages**

Disadvantages of Class I direct composite restoration as follows:

1. Polymerization shrinkage effects.
2. Lower fracture toughness than most indirect restorations.
3. More technique-sensitive than amalgam restorations and some indirect restorations.
4. Possible greater localized occlusal wear.

#### ***Pit-and-Fissure Sealants***

Pits and fissures typically result from an incomplete coalescence of enamel and are particularly prone to caries. These areas can be sealed with a low-viscosity fluid resin after acid-etching. Long-term clinical studies indicate that pit and fissure sealants provide a safe and effective method of



preventing caries. In children, sealants are most effective when they are applied to the pits and fissures of permanent posterior teeth immediately on eruption of the clinical crowns, provided proper isolation can be achieved. Adults also can benefit from the use of sealants if the individual experiences an increase in caries susceptibility because of a change in diet, lack of adequate saliva, or a particular medical condition. Most currently used sealant materials are light-activated urethane dimethacrylate or BIS-GMA (bisphenol A-glycidylmethacrylate) resins.

### ***Clinical Technique for Class I Direct Composite Restorations***

#### ***Tooth Preparation***

As a general rule, the tooth preparation for direct posterior composites involves:

- 1) creating access to the faulty structure,
- 2) removal of faulty structures (caries, defective restoration),
- 3) creating convenience form for the restoration. Retention is obtained by bonding.

#### ***Small to Moderate Class I Direct Composite restorations***

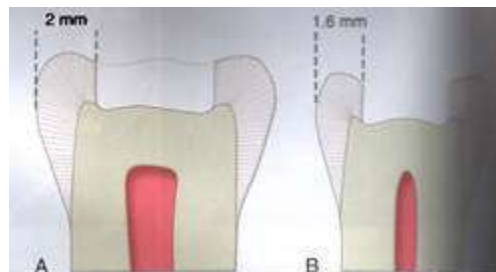
Small to moderate Class I composite restorations may use minimally invasive tooth preparations and do not require typical resistance and retention form features. Instead, these conservative preparations typically use more flared cavosurface forms without uniform or flat pulpal or axial walls. These preparations are less specific in form, having a scooped-out appearance. They are prepared with a small round or elongated pear diamond or bur with round features.

The initial pulpal depth is approximately 0.2 mm inside the DEJ but may not be uniform (i.e., the pulpal floor is not flat throughout its length). Usually, a more rounded, and perhaps smaller, cutting instrument is used for this preparation, in an attempt to be as conservative as possible in the removal of the tooth structure. If a round instrument is used, the resulting cavosurface margin angle may be more flared (obtuse) than if an elongated pear instrument is used.

Various cutting instruments may be used for Class I preparations; the size and shape of the instrument generally dictated by the size of the lesion or other defect or type of defective restoration being replaced. Both diamond and diamond instruments can be used effectively. It be noted that diamond instruments create a thicker smear layer, however, which might make bonding more difficult self-etch bonding systems.

The objective of the tooth preparation is to remove all of the caries or fault as conservatively as possible. Because the composite is bonded to the tooth structure, other less involved, or at-risk, areas can be sealed as part of the conservative preparation techniques. Sealants may be combined with the Class I composite restoration.

In large composite restorations, the tooth is entered in the area most affected by caries, with the elongated pearl diamond or bur positioned parallel to the long axis of the crown. When it is anticipated that the entire mesiodistal length of a central groove will be prepared, it is easier to enter the distal portion first and then transverse mesially. This technique permits better vision to the operator during the preparation. The pulpal floor is prepared to an initial depth that is approximately 0.2 mm internal to the DEJ. The instrument is moved mesially, following the central groove, and any fall and rise of the DEJ. Mesial, distal, facial, and lingual extensions are dictated by the caries, old restorative material, or defect, always using the DEJ as a reference for both extensions and pulpal depth. The cuspal and marginal ridge areas should be preserved as much as possible. Although the final bonded composite restoration would help restore some of the strength of weakened, unprepared tooth structure, the outline form should be as conservative as possible. Extensions toward cusp tips should be as minimal as possible. Extensions into marginal ridges should result in at least 1.5 mm of remaining tooth structure (measured from the internal extension to the proximal height of contour) for premolars and approximately 2 mm for molars. These limited extensions help preserve the dentinal support of the marginal ridge enamel and cusp tips. As the instrument is moved along the central groove, the resulting pulpal floor is usually moderately flat (as a result of the shape of the tip of the instrument) and follows the rise and fall of the DEJ. If extension is required toward the cusp tips, the same depth that is approximately 0.2 mm inside the DEJ is maintained, usually resulting in the pulpal floor rising occlusally. The same uniform depth concept also is appropriate when extending a facial or lingual groove radiating from the occlusal surface. When a groove extension is through the cusp ridge, the instrument prepares the facial (or lingual) portion of the faulty groove at an axial depth of 0.2 mm inside the DEJ and gingivally to include all caries and other defects.



*Figure 26. Mesiodistal extension.  
Preserve dentin support of marginal ridge enamel  
A, Molar. B, Premolar.*

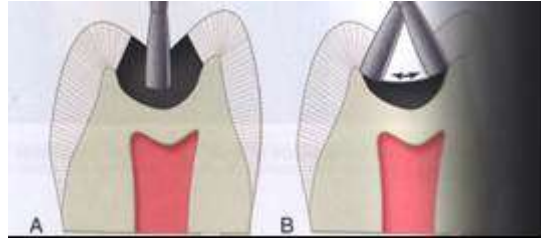


Figure 27. A, After initial entry cut at correct initial depth (1,5 mm), the caries remains facially and lingually.  
B, Orientation of diamond must be tilted as the instrument is extended facially or lingually to maintain a 1,5 mm depth.

After extending the outline form to sound tooth structure, if any caries or old restorative material remains on the pulpal floor, it should be removed with the appropriately-sized round bur or hand instrument. The occlusal margin is left as prepared. No attempt is made to place additional beveling on the occlusal margin because it may result in thin composite in areas of heavy occlusal contact. Because of the occlusal surface enamel rod direction, the ends of the enamel rods already are exposed by the preparation, which further reduces the need for occlusal bevels.



Figure 28. Directions of enamel rods

### ***Restorative technique. Insertion and Light-Activation of the Composite***

Composite insertion hand instruments or a compule may be used to insert the composite material. The dispenser, for example, a syringe or compule, must be kept covered when not in use to prevent premature hardening of the material. Small increments of composite material are added and successively light-activated. It is important to place (and light-activate) the composite incrementally to maximize the polymerization depth of cure and possibly to reduce the negative effects of polymerization shrinkage.

The term "configuration factor" or "C-factor" has been used to describe the ratio of bonded to unbonded surfaces in a tooth preparation and restoration. A typical Class I tooth preparation will have a high C-factor of 5 (five bonded - pulpal, facial, lingual, mesial, and distal-vs. one unbonded surface-occlusal). The higher the C-factor of a tooth preparation, the higher the potential for composite polymerization shrinkage stress, as the composite shrinkage deformation is restricted by the bonded surfaces. Incremental

insertion and light-activation of the composite may reduce the negative C-factor effects for Class I composite restorations.

### **Tests to the topic**

#### **1. Point the main requirements for the formation of the classical class I cavities by Black:**

- a. Bottom is flat.
- b. Cavity walls are at an acute angle to the bottom.
- c. Walls are vertical.
- d. The right angle between the bottom and the walls.
- e. Bottom is convex.

#### **2. What are the peculiarities of class I cavity preparation by Black with deep caries:**

- a. The formation of retention points.
- b. Softened dentin may be remained at the bottom.
- c. Necessarily steep wall.
- d. Pigmented dentin may be remained at the bottom.
- e. The bottom can be a relief.
- f. Pins are used.

#### **3. What are the peculiarities of class I cavities preparation using composite materials?**

- a. Mesial-distal walls are parallel to the axis of the tooth or have a slight expansion of the occlusion (<10 degrees).
- b. Bottom is flat.
- c. Bucco-lingual walls are parallel to the axis of the tooth or slightly converge (<10 degrees).
- d. The angles between the bottom and walls of the cavity are rounded.
- e. The angle between the bottom and walls of the cavity are right.

#### **4. What are the advantages of amalgam?**

- a. High compressive strength.
- b. Ease of use.
- c. Excellent wear resistance.
- d. Favorable long-term clinical research results.
- e. Lower cost than composite restoration.
- f. Chemically resistant.
- g. Esthetic.

#### **5. What are the disadvantages of amalgams?**

- a. Non-esthetic.

- b. Require increased tooth structure removal during tooth preparation.
- c. Require insulating layers.
- d. Initial marginal leakage.
- e. High thermal conductivity.
- f. Easy to form a seal.

**6. What are the indications for the use of amalgams?**

- a. Class III cavities.
- b. Class I cavities.
- c. Class V cavities.
- d. Class II cavities.
- e. Class IV cavity.

**7. What are the advantages of composites?**

- a. Aesthetics.
- b. Low thermal conductivity.
- c. Not toxic.
- d. Strengthening tooth structure.
- e. Good polishing.

**8. What complications polymerization shrinkage causes?**

- a. Secondary caries.
- b. Marginal gap formation between the restoration and the tooth surface.
- c. Violation of marginal fit.
- d. Microcracks.
- e. Microleakage.
- f. Hypersensitivity.
- g. Discoloration of seals.

**9. In what time is recommended to make a final finishing and polishing amalgam fillings?**

- a. In 2 hours.
- b. In 4 hours.
- c. In 12 h.
- d. In 24 h.

**10. What are the advantages of glass ionomer cements?**

- a. Chemical adhesion.
- b. Coefficient of thermal expansion close to that of hard tissue.
- c. The content of fluoride ions.
- d. All of the above.

## LESSON 13. DIAGNOSIS AND TREATMENT OF INTERPROXIMAL SURFACES CARIES OF THE GROUP OF POSTERIOR TEETH. THE CHOICE OF FILLING MATERIAL

The questions to be studied for the learning of the topic:

1. Locations lesions of Class II. Physiological role of proximal contact point.
2. Main and additional methods of diagnosis of carious lesions of Class II.
3. Principles of preparation of Class II carious lesions.
4. Peculiarities of preparation of Class II carious lesions for composite filling materials.
5. The methods of re-establishment of proximal contact point.
6. Matrix system for re-establishment of proximal contact point.

### Question 1. Locations lesions of Class II. Physiological role of proximal contact point.

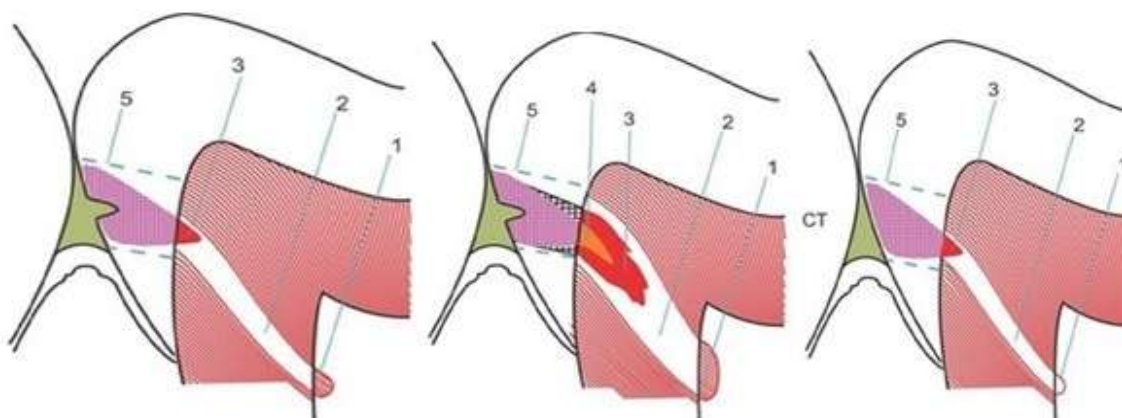


Figure 29. Schematic diagram of approximal caries progression

1 - tertiary or reparative dentine, 2 - dentine tubules, 3- affected layer of carious dentine, 4-infected layer of carious dentine, 5- enamel lesion.

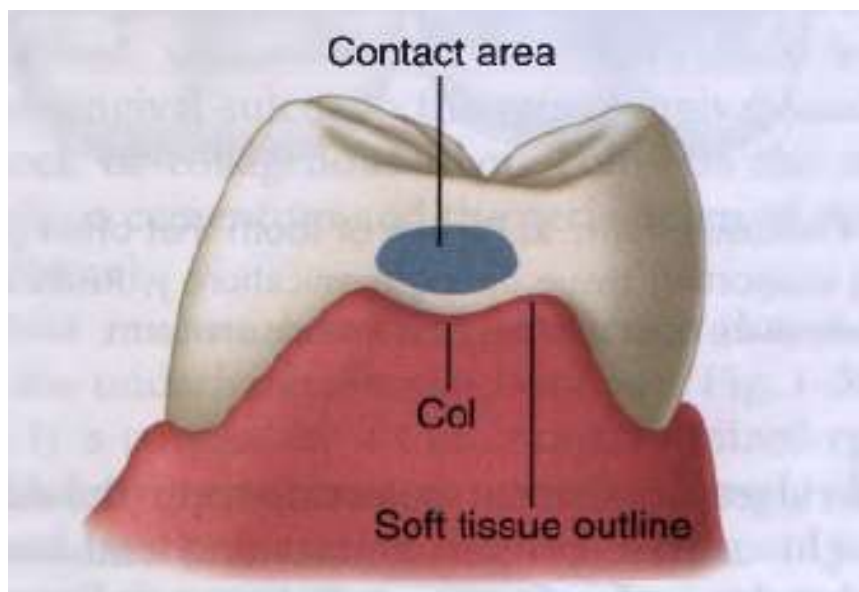
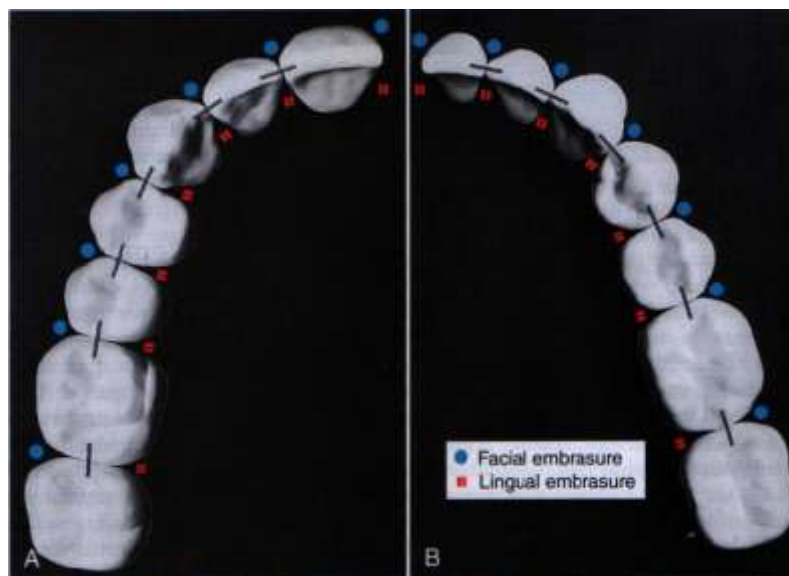
Lesions of Class II occur on the proximal surfaces of the posterior teeth - molars and premolars.

**Proximal Contact Area.** When teeth erupt to make proximal contact with previously erupted teeth, initially a contact point is present. The contact point increases in size to become a proximal contact area as the two adjacent tooth surfaces abrade each other during physiologic tooth movement.

The proximal contact area is located in the incisal third of the approximating surfaces of maxillary and mandibular central incisors. It is positioned slightly facial to the center of the proximal surface faciolingually.

Proceeding posteriorly from the incisor region through all the remaining teeth, the contact area is located near the junction of the incisal (or occlusal) and middle thirds or in the middle third.

Proximal contact areas typically are larger in the molar region, which helps prevent food impaction during mastication.



*Figure 30. Relationship of ideal interdental papilla to molar contact area.*

Adjacent surfaces near the proximal contacts (embrasures) usually have remarkable symmetry.

Initially, the interdental papilla fills the gingival embrasure. When the form and function of teeth are ideal and optimal oral health is maintained, the interdental papilla may continue in this position throughout life. When the gingival embrasure is filled by the papilla, trapping of food in this region is prevented.

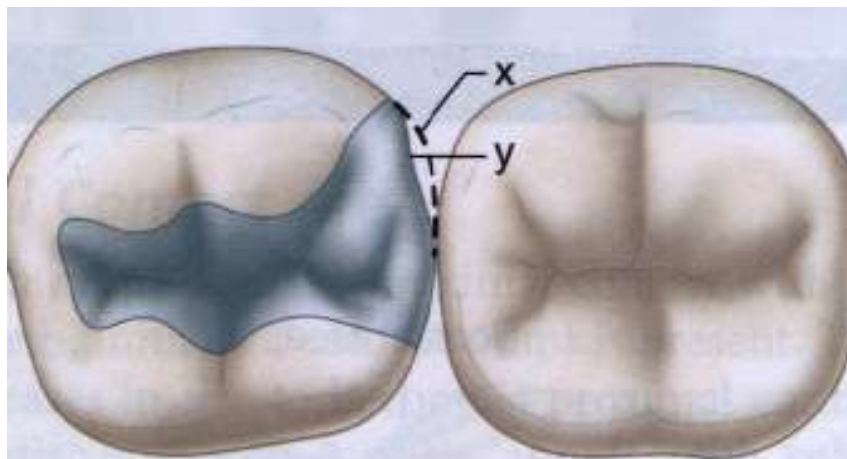
In a faciolingual vertical section, the papilla has a triangular shape between anterior teeth, whereas in posterior teeth, the papilla may be shaped



like a mountain range, with facial and lingual peaks and the col (“valley”) lying beneath the contact area. This col, a central faciolingual concave area beneath the contact, is more vulnerable to periodontal disease from incorrect contact and embrasure form because it is covered by nonkeratinized epithelium. The physiologic significance of properly formed and located proximal contacts and associated embrasures cannot be overemphasized; they promote normal healthy interdental papillae filling the interproximal space.

Improper contacts can result in food impaction between teeth, potentially increasing the risk of periodontal disease, caries, and tooth movement. In addition, retention of food is objectionable because of its physical presence and the halitosis that results from food decomposition. Proximal contacts and interdigitation of teeth through occlusal contacts stabilize and maintain the integrity of the dental arches.

The correct relationships of embrasures, cusps to sulci, marginal ridges, and grooves of adjacent and opposing teeth provide for the escape of food from the occlusal surfaces during mastication. When an embrasure is decreased in size or absent, additional stress is created on teeth and the supporting structures during mastication. Embrasures that are too large provide little protection to the supporting structures as food is forced into the interproximal space by an opposing cusp. A prime example is the failure to restore the distal cusp of a mandibular first molar when placing a restoration.



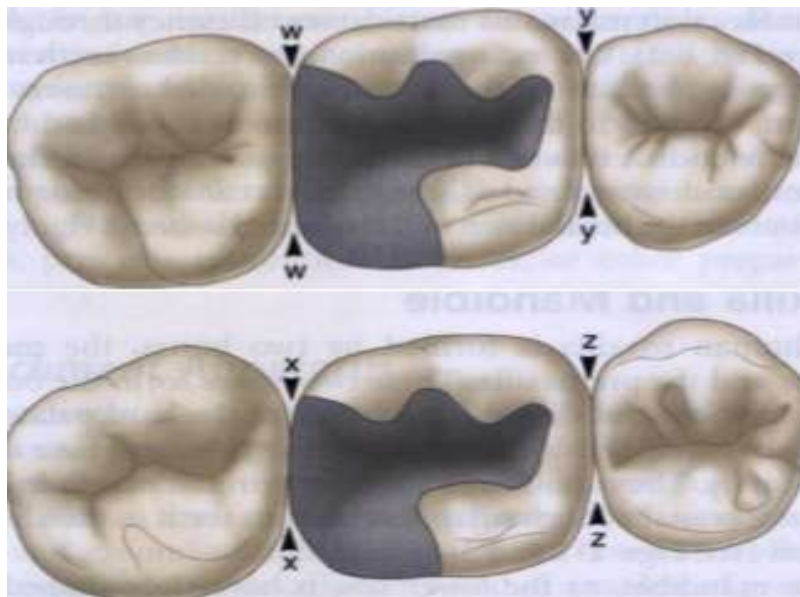
*Figure 31. Embrasure form, x, Portion of tooth that offers protection to underlying supporting tissue during mastication, y. Restoration fails to establish adequate contour for good embrasure form*

Lingual embrasures are usually larger than facial embrasures and this allows more food to be displaced lingually because the tongue can return the food to the occlusal surface more easily than if the food is displaced facially into the buccal vestibule. The marginal ridges of adjacent posterior teeth should be at the same height to have proper contact and embrasure forms.



When this relationship is absent, it causes an increase in the problems associated with weak proximal contacts and faulty embrasure forms.

Preservation of the curvatures of opposing cusps and surfaces in function maintains masticatory efficiency throughout life. Correct anatomic form renders teeth more self-cleansing because of the smoothly rounded contours that are more exposed to the cleansing action of foods and fluids and the frictional movement of the tongue, lips, and cheeks. Failure to understand and adhere to correct anatomic form can contribute to the breakdown of the restored system.

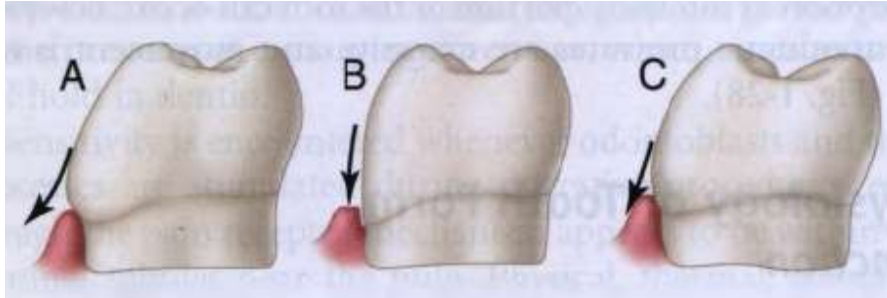


*Figure 32. Embrasure form, w, improper embrasure form caused by overcontouring of restoration resulting in unhealthy gingiva from lack of stimulation, x, Good embrasure form, y, Frictional wear of contact area has resulted in decrease of embrasure dimension, z, When the embrasure form is good, supporting tissues receive adequate stimulation from foods during mastication*

**Contours.** Facial and lingual surfaces possess a degree of convexity that affords protection and stimulation of supporting tissues during mastication. The convexity generally is located at the cervical third of the crown on the facial surfaces of all teeth and the lingual surfaces of incisors and canines. The lingual surfaces of posterior teeth usually have their height of contour in the middle third of the crown. Normal tooth contours act in deflecting food only to the extent that the passing food stimulates (by gentle massage) and does not irritate supporting tissues. If these curvatures are too

great, tissues usually receive inadequate stimulation by the passage of food. Too little contour may result in trauma to the attachment apparatus.

These tooth contours must be considered in the performance of operative dental procedures. Improper location and degree of facial or lingual convexities can result in serious complications, in which the proper facial contour is disregarded in the placement of a cervical restoration on a mandibular molar.



*Figure 33. Contours. Arrows show pathways of food passing over facial surface of mandibular molar during mastication.*

*A, Over-contour deflects food from gingiva and results in under-stimulation of supporting tissues.*

*B, Under-contour of tooth may result in irritation of soft tissue.*

*C, Correct contour permits adequate stimulation for supporting tissue, resulting in healthy condition*

Over-contouring is the worst offender, usually resulting in increased plaque retention that leads to a chronic inflammatory state of the gingiva. The proper form of the proximal surfaces of teeth is just as important to the maintenance of periodontal tissue as is the proper form of facial and lingual surfaces. The proximal height of contour serves to provide: (1) contacts with the proximal surfaces of adjacent teeth, thus preventing food impaction, (2) adequate embrasure space apical to the contacts for gingival tissue, supporting bone, blood vessels, and nerves that serve the supporting structures.

## **Question 2. Main and additional methods of diagnosis of carious lesions of Class II.**

*The main methods of diagnosis:*

### **1. Questioning:**

- Complaints.
- Patient history.
- Medical history.

### **2. Clinical Examination:**

- Probing:
- To determine of roughness of enamel surface.

- To determine of dentin density (soft or hard) at a bottom and a walls of caries cavity.
- To determine of sensitivity (a bottom and a walls of caries cavity).
- Drying.
- Reaction on thermal agents (cold, heat).

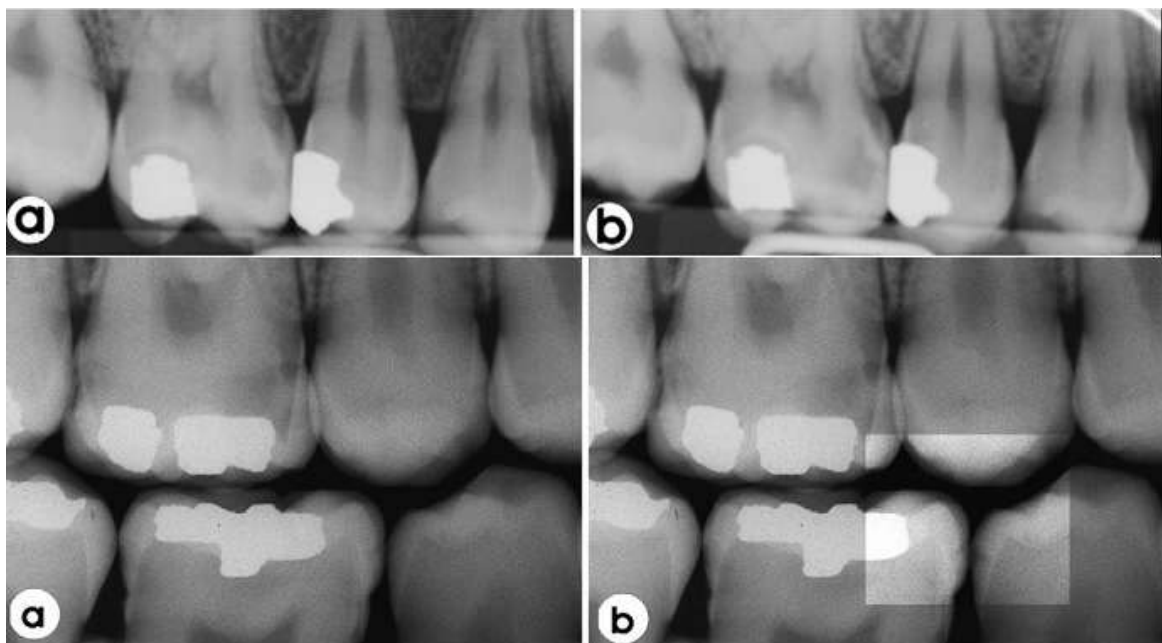
*The additional methods of diagnosis of carious lesions of Class II.*

- ✓ Radiologic and clinical examination.
- ✓ Emerging diagnostic techniques.
- ✓ Fluorescence.
- ✓ Fiber-optic transillumination (FOTI).
- ✓ Digitally imaging fiber-optic transillumination (DIFOTI).
- ✓ Electrical conductivity.

Radiographic examination is useful to confirm the extent of caries, to detect lesions where visual examination of the tooth surface is hampered and to serve as an aid in making appropriate clinical decisions.

**Radiography.** The most commonly used radiographic method for detecting caries lesions is the bitewing technique. It is meant to find lesions that are hidden from a clinical visual examination, such as when a lesion is hidden by an adjacent tooth, proximal caries.

**Bitewing radiography** allows accessibility to surfaces that may not be seen in the clinical visual–tactile examination, and allows the depth of lesions to be assessed. Other advantages are that it is not invasive, and does not damage tooth structure like an incorrectly used dental probe might. Radiographs can also be filed and reexamined at a later date to compare with a more recent image to detect whether a lesion is progressing or not.



*Figure 34. Approximal caries on bitewing radiographs.*

**Digital radiography** – which is increasingly replacing bitewing radiography and that is as accurate as film for the detection of caries lesions.

**Digital image enhancement** – which studies show can provide superior results to radiographs when enhanced correctly but takes a significant amount of technical skill.

**Digital subtraction radiography** – which is not typically used in a clinical setting, also because of the high level of technical skill needed to perform correctly.

**Tuned aperture computed tomography** – which shows improved diagnostic accuracy in caries lesion detection, but with equipment that is too expensive for most clinical practices.

### **Question 3. Principles of preparation of Class II carious lesions.**

If the cavity is localized below the equator of the teeth, cavity should be prepared, as a Class 5 cavity (with the possibility of access). Additional box is formed on the vestibular surface.

If the cavity is located on the equator or higher, it is required to prepare the box on the occlusal surface. The bottom of the proximal box is vertically. Gingival wall can be formed not only by direct, but at an acute angle to the bottom of the cavity. This prevents injuries of the gingiva and improves the retention of the filling.

In a place of convergence of the gingival wall and of the cavity bottom the corner slightly rounded in order to avoid stress of hard dental tissues.

The occlusal box has the following parameters:

- ✓ The bottom is perpendicular to the proximal box.
- ✓ The length is 1/2 - 1/3 of the mesiodistal size of the teeth
- ✓ The width is equal to the width of the proximal box.
- ✓ The depth is a little deeper enamel-dentine connection.

If there are carious cavities of both interproximal surfaces, it is perhaps to combine of these cavities.

In order to improve the fixation of the seal it is necessary to perform retention points in the dentin in the gingival area.

### **Question 4. Peculiarities of preparation of class II carious lesions for composite filling materials.**

**The tooth preparation** for Class II direct composites involves:

- 1) creating access to the faulty structure,
- 2) removal of faulty structures (caries, defective restoration and base material, if present),
- 3) creating the convenience form for the restoration. Retention, use mechanical retention features in the tooth preparation Class II composite restorations.

***Small Class II direct composite restorations*** are often u primary caries lesions, that is, initial restorations. A round or elongated pearl diamond or bur with round features may be used for this preparation to scoop out the carious or faulty material from the occlusal and proximal surfaces. The pulpal and axial depths are dictated only by the depth lesion and are not uniform. The proximal extensions likewise are dictated only by the extent of the lesion but may require the use another instrument with straight sides to prepare walls 90 degrees or greater. The objectives are to remove caries or the defect conservatively and remove friable tooth structure.

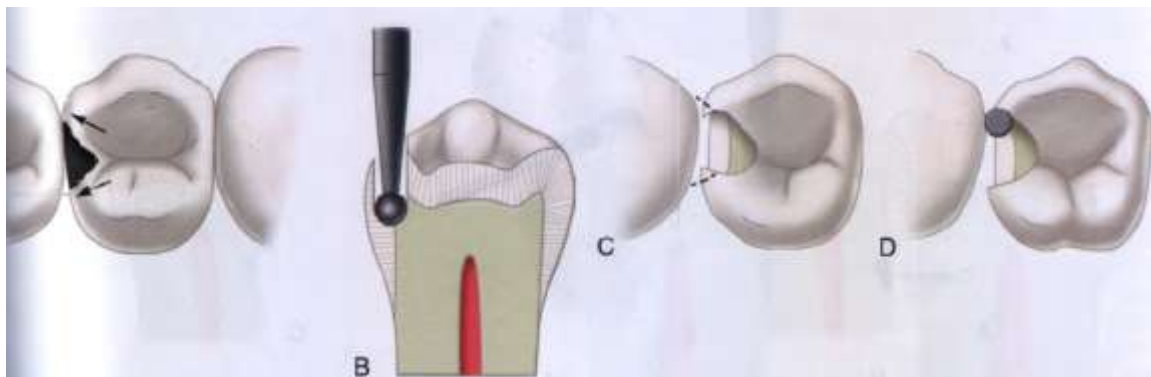


Figure 35. A, Pre-operative visualization of faciolingual proximal box extensions. Arrows indicate desired extensions. B, Round or oval, small elongated pearl instrument used. C and D, Facial, lingual, and gingival margins may need undermined cavosurface enamel (endicated by dotted lines) removed with straight-sided thin and flat-tipped rotary instrument or hand instrument

Another conservative design for small Class II composites is the box only tooth preparation.

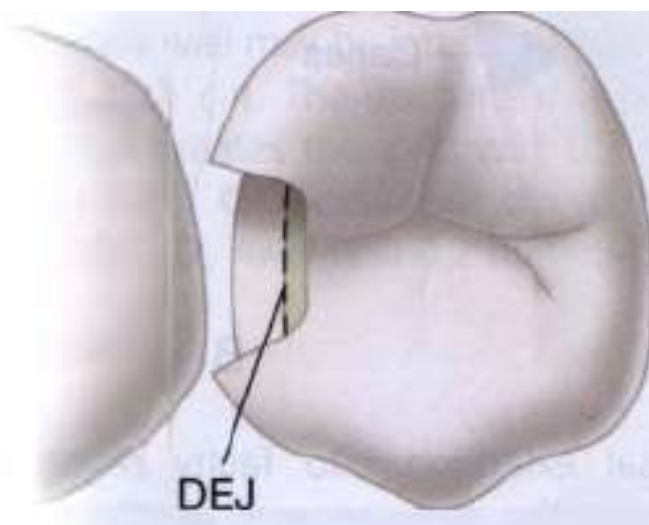
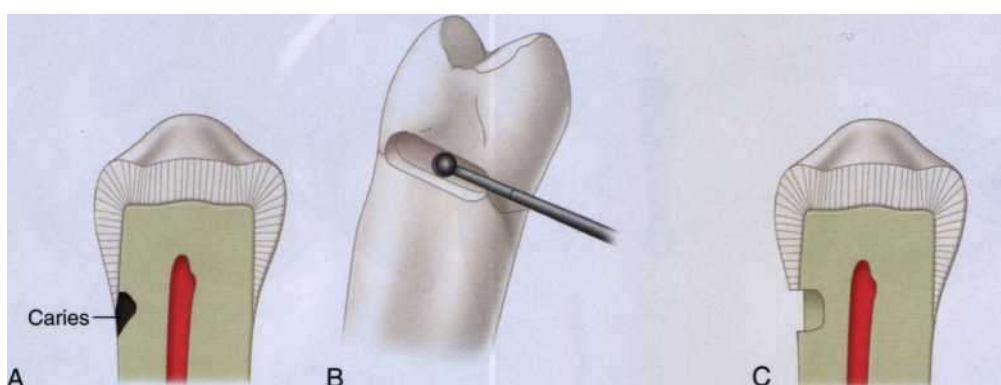


Figure 36. Box-only Class II composite preparation.

This design is indicated when only the proximal surface is defective, with no lesions on the occlusal surface. A proximal box is prepared elongated pear or round instrument, held parallel to the long axis of the tooth crown. The instrument is extended through marginal ridge in a gingival direction. The axial depth dictated by the extent of the caries lesion or fault. The form of the box depends on which instrument shape is used the more boxlike with the elongated pear and the more scooped with the round. The facial, lingual, and gingival extensions are dictated by the defect or caries. No beveling or secondary retention is indicated.

A third conservative design for restoring proximal lesions on posterior teeth is the facial or lingual slot preparation.



*Figure 37. Facial or lingual slot preparation. A, Cervical caries on the proximal surface. B, The round diamond or bur enters the tooth from accessible embrasure, oriented to the occlusogingival middle of the lesion. C, Slot preparation*

Here, a lesion is detected on the proximal surface, but the operator believes that access to the lesion can be obtained from either a facial direction or a lingual direction, rather than through marginal ridge in a gingival direction. Usually, a small diamond or bur is used to gain access to the lesion. The instrument is oriented at the correct occluso – gingival position, and the entry is made with the instrument adjacent tooth as possible, preserving as much of the facial or lingual surface as possible. The preparation is extended occlusally, facially, and gingivally enough to remove the lesion. The axial depth is determined by the extent of the lesion. The occlusal, facial, and gingival cavosurface margins are 90 degrees or greater. Care should be taken not to undermine the marginal ridge during the preparation.

### ***Moderate to Large Class II Direct Composite Restorations.***

The tooth preparation for moderate to large Class II direct composite restorations has features that resemble a more traditional Class II tooth preparation (amalgam) and include an occlusal step and a proximal box.



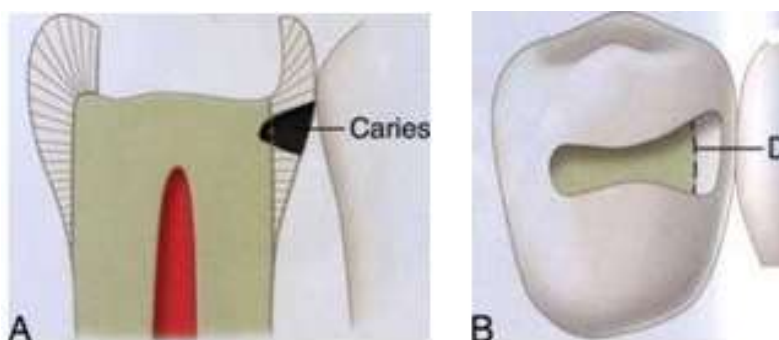
### ***Occlusal step***

The occlusal portion of the Class II preparation is prepared similarly as for the Class I preparation. The primary differences are related to technique of incorporating the faulty proximal surface. Pre-operatively, the proposed facial and lingual proximal extensions should be visualized.

Initial occlusal extension toward the involved proximal surface should go through the marginal ridge area at initial pulpal floor depth, exposing the DEJ. The DEJ serves as a guide for preparing the proximal box portion of the preparation.

When only one proximal surfaces is affected, the opposite marginal ridge should be maintained. The pulpal floor is prepared with the instrument to a depth that is approximately 0.2 mm inside the DEJ. The instrument is moved to include caries and all defects facially or lingually or both, as it transverses the central groove. Every effort should be made, however, to keep the faciolingual width of the preparation as narrow as possible. The initial depth is maintained during the mesiodistal movement, but follows the rise and fall of the underlying DEJ.

The pulpal floor is relatively flat in a faciolingual plane but may rise and fall slightly in a mesiodistal plane.



*Figure 38. Occlusal extension into faulty proximal surface. A and B, Extension exposes the dentinoenamel junction (DEJ) but does not hit adjacent tooth. Facial and lingual extensions as preoperatively visualized*

If caries remains in dentin, it is removed after preparation outline, including the proximal box extensions, has been established.

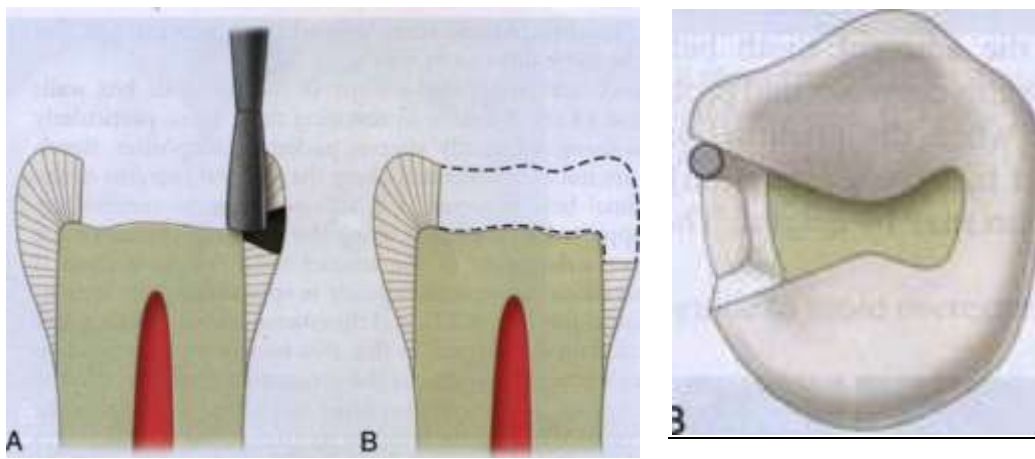
Because the facial and lingual proximal extensions of the faulty proximal surface were visualized preoperatively, the occlusal extension toward that proximal surface begins to widen facially and lingually to begin to outline those extensions as conservatively as possible. Care is taken to preserve cuspal areas as much as possible during these extensions. At the same time, the instrument extends through the marginal ridge to within 0.5 mm of the outer contour of the marginal ridge. This extension exposes the proximal DEJ and protects the adjacent tooth. At this time, the occlusal

portion of the preparation is complete except for possible additional pulpal floor caries excavation. The occlusal walls generally converge occlusally because of the inverted s the instrument.

### ***Proximal box***

Typically, caries develops on a proximal surface immediately gingival to the proximal contact. The extent of the caries lesions and amount of old restorative material are two factors that dictate the facial, lingual, and gingival extensions of the proximal box of the preparation. Although it is not required to extend the proximal box beyond contact with the a tooth (i.e., provide clearance with the adjacent tooth), it may simplify the preparation, matrix placement, and contouring procedures. If all of the defect can be removed without extending the proximal preparation beyond the contact, however, the restoration of the proximal contact with the composite is simplified.

Before the instrument is extended through the marginal ridge, the proximal ditch cut is initiated. The operator holds the instrument over the DEJ with the tip of the instrument positioned to create a gingivally directed cut that is 0,2 mm inside the DEJ. For a № 245 instrument with a tip diameter of 0.8 mm, this would require one-fourth of the instrument's tip positioned over the dentin the side of the DEJ (the other three fourths of the tip over the enamel side). The instrument is extended facially, lingually and gingivally to include all of the caries or old material, or both.



*Figure 39. Using a smaller nstrument to prepare the cavosurface margin and lingual proximal walls.*

The faciolingual cutting motion follows the DEJ and is usually slightly convex arc outward.

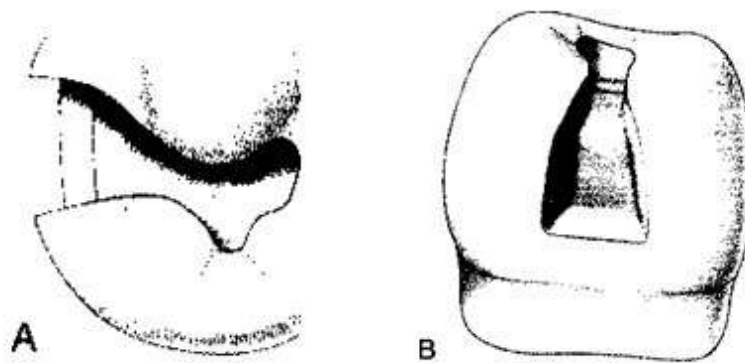
During this entire cutting, the instrument is held parallel to the long axis of the tooth crown. The facial and lingual margins are extended as necessary and should result in at least a 90 degree margin, more obtuse being acceptable as well. If the preparation is conservative, a smaller, thinner



instrument is used to complete the faciolingual wall formation, avoiding the adjacent tooth. Alternatively, a sharp hand instrument such as a chisel, hatchet, or a gingival margin trimmer can be used to finish the enamel wall. At this point, the remaining proximal enamel that was initially maintained to prevent damage to the adjacent tooth has been removed. The gingival floor is prepared flat (because of the tip instrument) with an approximately 90-degree cavosurface margin. Gingival extension should be as minimal as possible, in an attempt to maintain an enamel margin. The axial wall should be 0.2 mm inside the DEJ and have a slight outward convexity. For large caries lesions, additional axial wall caries excavation may be necessary later, during final tooth preparation.

Proximal extension. The enamel margin on the gingival floor is critical for bonding, so it should be preserved, if not compromised. Any remaining infected dentin on the axial wall (or the pulpal floor) is excavated as part of the final tooth preparation.

If no carious dentin or other defect remains, the preparation is considered complete at this time. Because the composite is retained in the preparation by micromechanical retention, no secondary preparation retention features are necessary. No bevels are placed on the occlusal cavosurface margins because these walls already have exposed enamel rod ends because of the enamel rod direction in this area. A bevel placed on an occlusal margin may result in thin composite on the occlusal surface in areas of potentially heavy contact. This feature also could result in fracture or wear of the composite in these areas. Beveled composite margins also may be more difficult to finish.



*Figure 40. Final Class II composite tooth preparation. A, Occlusal view. B, Proximal view.*

Bevels are rarely used on any of the proximal box walls because of the difficulty in restoring these areas, particularly when using inherently viscous packable composites. Bevels also are not recommended along the gingival margins of the proximal box; however, it is still necessary to remove any

unsupported enamel rods along the margins because of the gingival orientation of the enamel rods. For most Class II preparations, this margin already is approaching the cemento-enamel junction (CEJ), and the enamel is thin. Care is taken to maintain any enamel in this area to achieve a preparation with all-enamel margins. If the preparation extends onto the root surface, more attention must be focused on keeping the area isolated during the bonding technique, but no differences in tooth preparation are required. When the gingival floor is on the root surface (no enamel at the cavo- surface margin), the use of a glass ionomer material may decrease microleakage and recurrent caries. Usually, the only remaining final tooth preparation procedure that might be necessary is additional excavation of carious on either the pulpal floor or the axial wall. If necessary, a round bur or appropriate spoon excavator is used to remove any remaining caries.

**Question 5. The methods of re-establishment of proximal contact point.**

**Restorative Technique. Matrix Application.**

One of the most important steps in restoring Class II preparations with direct composites is the selection and proper placement of the matrix. In contrast to amalgam, which can be condensed to improve the proximal contact, Class II composites are almost totally dependent on the contour and position of the matrix for establishing appropriate proximal contacts. Early wedging and re-tightening of the wedge during tooth preparation aid in achieving sufficient separation of teeth to compensate for the thickness of the matrix band. Before placing the composite material, the matrix band must be in absolute contact with (touching) the adjacent contact area.

Generally, the matrix is applied before adhesive placement. An ultra-thin metal matrix band generally is preferred for the restoration of a Class II composite because it is thinner than a typical metal band and can be contoured better than a clear polyester matrix. No significant problems are experienced in placing and light-activating composite material when using a metal matrix as long as small incremental additions (2 mm each or less) are used.

Although a Tofflemire-type matrix can be used for restoring a two-surface tooth preparation, pre-contoured sectional metallic matrices are preferable, because only one thickness of metal matrix material is encountered instead of two, making contact generation easier.

These sectional matrices are relatively easy to use, very thin, and come in different sizes that can be used according to the clinical situation. There are several systems available, and selection is based on operator preference. These systems may use a bitine ring:

- 1) aid in stabilizing the matrix band.
- 2) provide additional tooth separation while the composite is inserted.

The primary benefit of these systems is a simpler method for establishing an appropriate composite proximal contour and contact. Use of these systems for restoring wide faciolingual proximal preparations requires careful application; otherwise, the bitine ring prongs may cause deformation of the band, resulting in a poor restoration contour.

When both proximal surfaces are involved, a Tofflemire retainer with an ultra-thin (0.001 inch), burnishable band is used. The band is contoured, positioned, wedged, shaped, as needed, for proper proximal contacts and embrasures. Before placement, the metal matrix band for po: composites should be burnished on a paper pad to proper proximal contour to the band (the same as a mfl for amalgam). Alternatively, an ultra-thin precontoured metal matrix band may be used in the Tofflemire retainer.

If the Tofflemire matrix band is open excessively along lingual margins of the preparation (usually because of the contour of the tooth), a “tinner’s joint” can be used to close the matrix band. This joint is made by grasping the lingual portion of the matrix band with No. 110 pliers and the band tightly together above the height of contour of the tooth.

***Insertion and Light-Activation of the Composite.*** Hand instruments or a «composite gun» may be used to insert the composite material. It is important to place (and light-activate) the composite incrementally to maximize the curing potential and to reduce negative effects of polymerization shrinkage. There are many techniques for the restoration of the proximal box. Research comparing different insertion and light-activation techniques is not conclusive, and no single technique has been universally accepted. The number of increments depend on the size of the proximal box.

Oblique incremental technique: the first increment(s) should be placed along the gingival floor and should extend slightly up the facial (or lingual) wall.

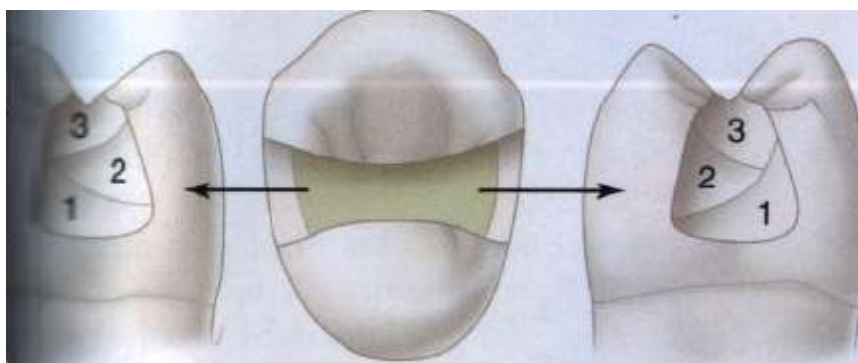


Figure 41. Directions of insertions of composite material

This increment (or increments for large box) should be only approximately 1 to 2 mm thick because it is the farthest increment from the

curing light and most critical in establishing a proper gingival seal. A second increment is then placed against the lingual (or facial) wall, to restore about two thirds of the box. The final increment is then placed to complete the proximal box and develop the marginal ridge. Subsequent additions, if needed, are made and light-activated (usually not exceeding 2 mm in thickness at a time) until the proximal box is fully restored.

Increments should be light-activated for as long as needed, depending on the shade and opacity of the composite used, the distance of the composite from the light tip, and the power of the unit. Regardless of the number of increments needed, when restoring the proximal box, an effort should be made to develop the anatomy of marginal ridge without excessive composite, to reduce the amount of cutting needed during contouring and polishing.

When the proximal box is completed, the occlusal step of the preparation is restored exactly as for the Class I direct composite restoration, that is, using an anatomic layering technique.

The incremental insertion and light-activation technique described provides enhanced control over the application and polymerization of individual increments of composite. The incremental technique also allows for (1) orientation of the polymerization light beam according to the position of each increment of composite, thus enhancing the curing potential; (2) intrinsic restoration characterization with darker or pigmented composites; and (3) sculpture of the restoration occlusal stratum with a more translucent material simulating the natural enamel. Tight proximal contacts can also be better achieved when composite is applied in increments. The matrix can be held in close contact with the adjacent proximal surface while the contact-related increment of composite is light-activated. A hand instrument with a large surface area (e.g., a small football-shaped or round-shaped burnisher) is well suited for that purpose. Once this increment is cured, the proximal contact is established, and remaining increments can be inserted and light-activated. The matrix is removed, and the restoration is cured from the facial and lingual directions. The restoration can be contoured and finished immediately after the last increment is cured.

When a stiffer or packable composite is used for the restoration of the proximal box, a very small increment of a flowable composite first in the proximal box can be used to improve marginal adaptation of the restoration.

***Contouring and Polishing of the Composite.*** Contouring can be initiated immediately after the composite material has been fully polymerized. If the occlusal anatomy was developed as described in the previous sections, the need for additional contouring is greatly minimized. If contouring is needed, the occlusal surface is shaped with a round or oval, 12-bladed carbide finishing bur or finishing diamond. Excess composite is removed at the proximal margins and embrasures with a flame-shaped, 12-

bladed carbide finishing bur or finishing diamond and abrasive discs. Narrow fin- strips may be used to smooth the gingival proximal surface. Care must be exercised in maintaining the position of the finishing strips gingival to the proximal contact area to avoid inadvertent opening of the contact. The occlusion is evaluated for proper contact. Further adjustments are made if needed, and the restorations are finished with appropriate polishing points, cups, brushes, or discs.

### **Technique for Extensive II Direct Composite restorations**

Direct composite is not usually indicated for extensive posterior restorations but may be indicated when economic and others factors prevent the patient from selecting a more expensive indirect restorations.

The primary differences for very large preparations includes the following:

- 1) some or all of the cusps may be capped;
- 2) extensions in most directions are greater,
- 3) secondary retention features are used;
- 4) more resistance form features are used.

A cusp must be capped if the operator believes it is likely to fracture if left in a weakened state. Cupping a cusp usually is indicated when the occlusal outline form extends more than two thirds the distance from a primary groove to a cusp tip. An operator sometimes may choose to ignore this general rule when using a bonded restoration if cusp will be capped as part of the preparation the subsequent indirect restoration.

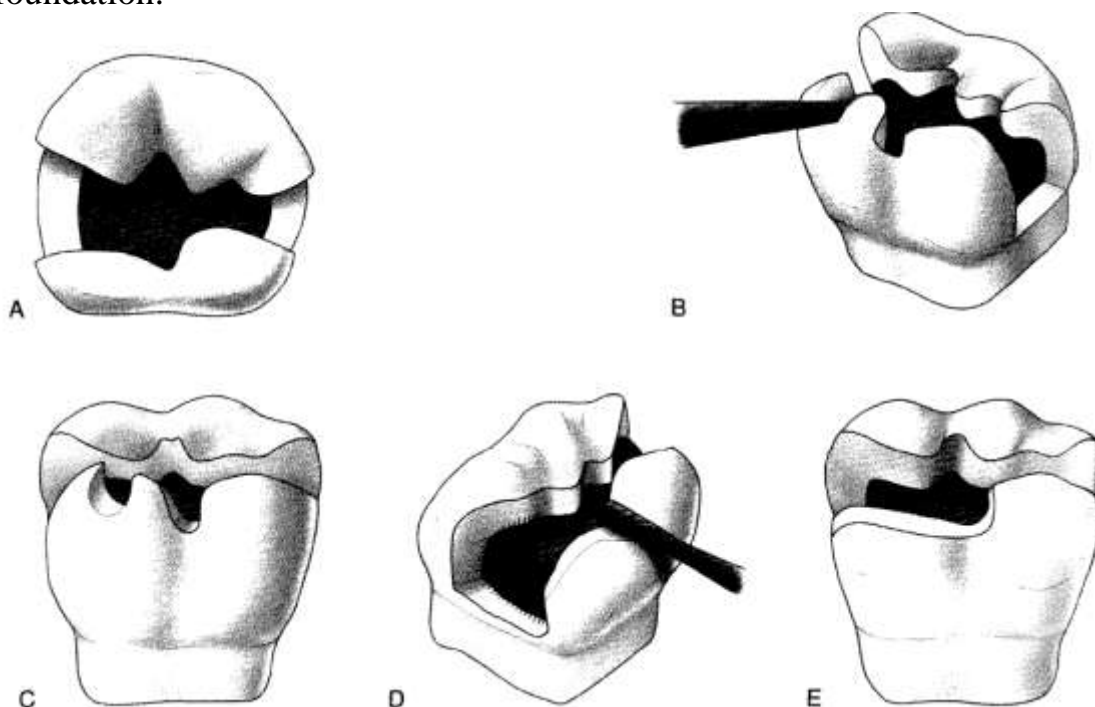
If the tooth has had endodontic treatment, the pulp chamber can be opened, and extensions can be made several millimeters into each treated canal. Because of the increased surface area for bonding and the mechanical retention from extensions into the canals, usually fewer secondary retention features are incorporated into the remaining tooth preparation.

**Tooth Preparation.** The elongated pear diamond or bur is used to prepare the occlusal step. As already indicated, the occlusal outline form is usually extensive. When moving the instrument from the central groove area toward a cuspal prominence, the pulpal depth that is approximately 0.2 mm inside the DEJ should be maintained, if possible. This creates a pulpal floor which rises occlusally as it is extended either facially or lingually. If a cusp must be capped, the side of the rotary instrument can be used first to make several depth cuts in the remaining cuspal form to serve as a guide for cusp reduction. Cusps should be capped as early in the tooth preparation procedure as possible, providing more access and visibility for the preparation. The depth cut is made with the instrument held parallel to the cuspal incline (from cusp tip to central groove) and approximately 1.5 to 2 mm deep. For a large cusp, multiple depth cuts can be made. Then, the instrument is used to join the depth cuts and extend to the remainder of the cuspal form. The reduced

cuspal surface has a relatively flat surface that may rise and fall with the normal mesial and distal inclines of the cusp. It also should provide enough clearance with the opposing tooth to result in approximately 1.5 to 2 mm of composite material to restore form and function. The cuspal reduction should be blended in with the rest of the occlusal step portion of the preparation.

The proximal boxes are prepared as described previously. The primary difference is that they may be much larger, that is, more extension in every direction. The extent of the lesion may dictate that a proximal box extend around the line angle of the tooth to include caries or faulty facial or lingual tooth structure. When the outline form has been established (the margins extended to sound tooth structure), caries at the pulpal and axial walls is excavated and the preparation is assessed carefully for additional retention form needs.

Retention form can be enhanced by the placement of grooves, locks, coves, or slots. All such retention form features must be placed entirely in dentin, not undermining and weakening any adjacent enamel. At times, bevels may be placed on available enamel margins to enhance retention form, even on occlusal areas. Retention form for foundations must be placed far enough inside the DEJ (at least 1 mm) to remain after the crown preparation is done subsequently. Otherwise, the potential retentiveness may be lost for the foundation.



*Figure 42. Cusp reduction. A, The initial outline form weakens the mesiolingual cusp enough to necessitate capping. B, Depth cuts made. C, cuts. D, Cusp reduction prepared. E, Vertical wall maintained between reduced and unreduced cusps.*

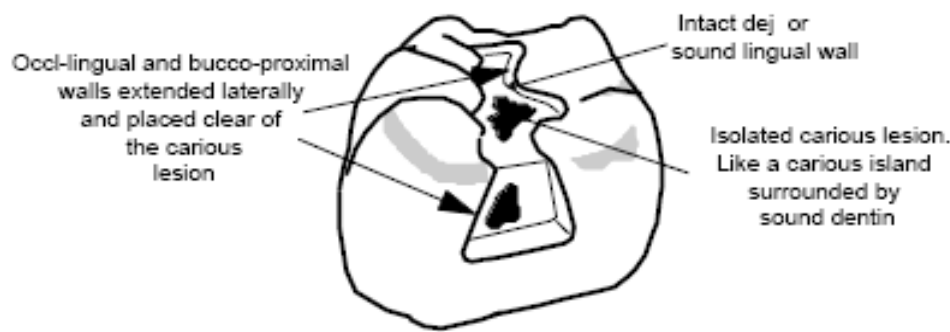


Figure 43. The bucco-proximal and occluso-lingual walls are extended laterally and placed 1/4 to 1/2mm clear of the carious lesion. The carious lesion is isolated and accessible for removal of the remaining carious dentin

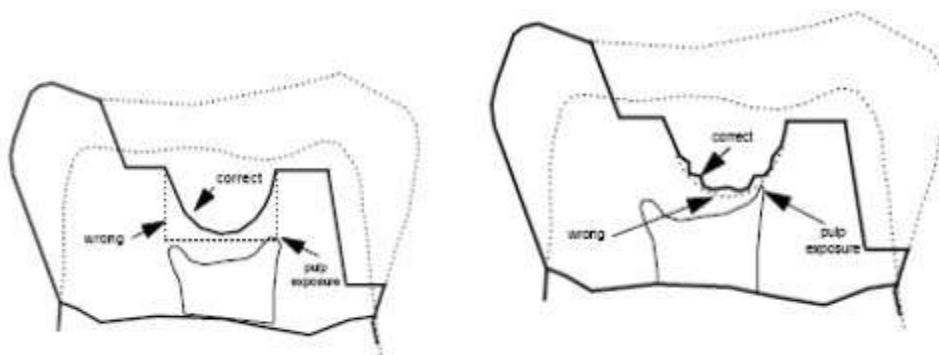


Figure 44. Correct and incorrect variants of preparations of cavity bottom.

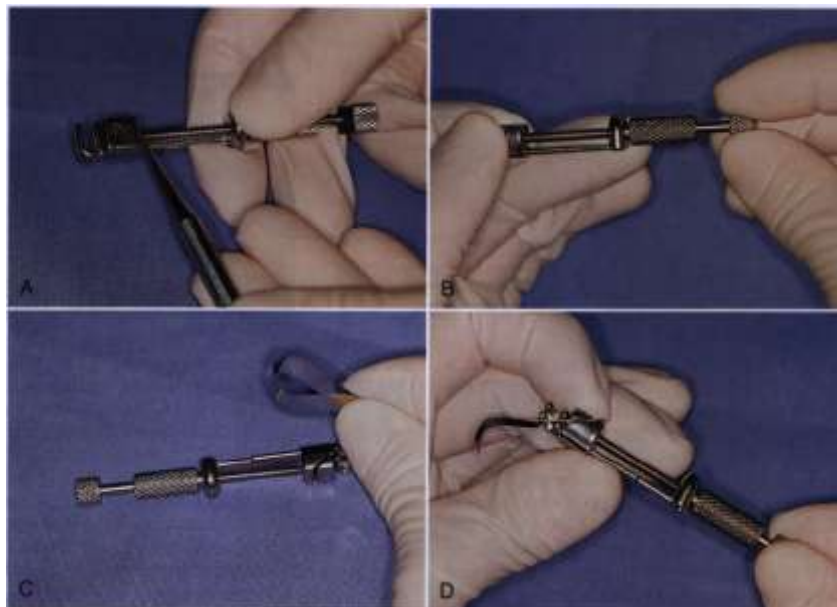
**Restorative Technique. Matrix Application.** Matrix placement is more demanding for these large restorations because more tooth structure is missing, and more margins may be subgingival. Proper burnishing of the matrix band to achieve appropriate axial contours is important, unless immediate full coverage of the tooth is planned. It also may be necessary to modify the matrix band to provide more subgingival extension in some areas and prevent extrusion of the composite from the matrix band-retainer tooth junction.

**Insertion and Light-Activation of the Composite.** When a light-activated composite is used, first it is placed in 1- to 2-mm increments into the most gingival areas of the proximal boxes. Each increment is cured, as directed. It may be helpful to use a hand instrument to hold the matrix against the adjacent tooth while light-activating the composite. This may assist in restoring the proximal contact.

Self-activated and dual-activated composite resin materials are frequently used for large composite foundations because these can be injected in the preparation in a single increment. However, it is recommended that even when dual-cured composites are used, they be carefully light-activated during after the final placement, as needed. When this technique used, the operator should carefully select the adhesive system, as some simplified

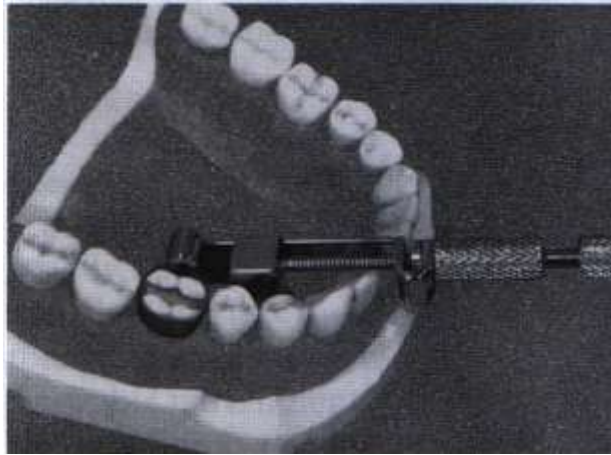
adhesives have been shown to be incompatible with some self-activated composite foundation materials. Acidic monomers in these adhesives scavenge the activators (tertiary amines) in the self-cure composite. If the activator does not function properly, the composite at the adhesive interface does not polymerize thoroughly and does not bond to the adhesive. Some manufacturers have introduced op chemical catalysts that can be mixed with the light adhesive to reduce or prevent this problem.

**Question 6. Matrix system for re-establishment of proximal contact point.**



*Figure 45. Straight and contra-angled Universal (Tofflemire) retainers. Bands with varying occlusogingival measurements are available.*





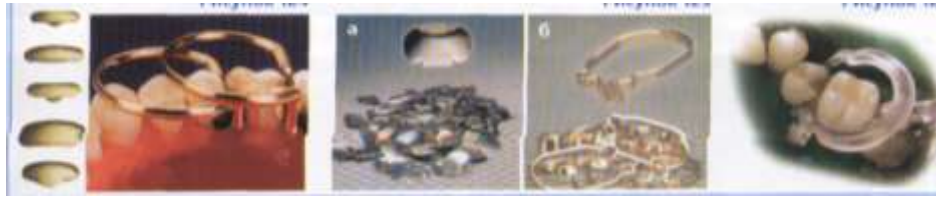
*Figure 46. Lingual positioning requires a contra-angled Universal retainer.*



*Figure 47. Precontoured bands for a Universal retainer*



*Figure 48. Contoured sectional matrices are ideal for posterior composite.*



*Figure 49. Hawe Adapt Sectional Matrix System (KerrHawe), Palodent (Dentsply).*



*Figure 50. Composite placement instruments with blunt cone-shaped ends are useful for placing increments of composite resin.*



*Figure 51. A large spoon makes a good contact-forming instrument.*

### **Tests to the topic**

#### **1. Locations of lesions of Class II include:**

- a. Occlusal surfaces of molars and premolars.
- b. Proximal surfaces of molars and premolars.
- c. Cutting edge.
- d. Lingual surface of anterior teeth.

#### **2. The role of contact proximal point:**

- a. Protect the papilla and periodontal tissues of the damage.
- b. Promotes the proper distribution of occlusal pressure.
- c. Preserves the integrity of dentition.
- d. All of the above.

**3. The most informative for the diagnosis of proximal caries are:**

- a. X-ray diagnosis.
- b. Electrotest.
- c. Transillumination;
- d. Dye method.

**4. Name a general rule of tooth preparation for direct posterior composite restoration:**

- a. Creating access to the faulty structure.
- b. Removal of faulty structures (caries, defective restoration and base material, if present).
- c. Creating the convenience form for the restoration.
- d. All of the above.

**5. What are criteria of final class ii composite tooth preparation?**

- a. The axial wall has a slight outward convexity.
- b. The gingival floor is prepared flat with approximately 90-degree cavosurface margin.
- c. The lingual and facial walls diverge to the proximal surface.
- d. The lingual and facial walls converge slightly to the occlusal surface.
- e. The lingual and facial margins are extended to 90-degree margin or more obtuse.
- f. All of the above.

**6. Is indicated beveling or secondary retention when preparation for small class ii direct composite restoration?**

- a. No.
- b. Yes.

**7. Name the primary differences of preparation technique for extensive class ii direct composite restoration:**

- a. Some or all of the cusps may be capped.
- b. Extensions in most directions are greater.
- c. Secondary retention features are used.
- d. More resistance form features are used.
- e. All of the above.

**8. What matrixe system are preferable for re-establishment of proximal contact point?**

- a. System of precontoured sectional metallic matrice.
- b. Tofflemire matrixe system.
- c. Universal retainer.

**9. What technique of insertion of composite provides control over the polymerization of composite?**

- a. Incremental technique.
- b. Technique of tunnel tooth preparation.
- c. Technique of insertion in single increment.

**10. What are in the gingival embrasure?**

- a. Interdental papilla.
- b. Gingival sulcus.
- c. Trapped food.
- d. Cement.

## LESSON 14. DIAGNOSTICS AND TREATMENT OF CERVICAL CARIES

The questions to be studied for the learning of the topic:

1. Cervical caries, definition.
2. Etiology of cervical caries.
3. Symptoms of cervical caries.
4. Diagnosis of cervical caries.
5. Treatment of cervical caries.
6. Prevention of cervical caries.

### **Question 1. Cervical caries, definition.**

Cervical caries is a special type of tooth decay which is characterized by destruction of teeth tissue at the cervical margin of the tooth. This disease progresses very rapidly permeating into the dental tissues and affecting the nerve canals. It is very difficult to detect this dental caries type at early stages and hard to manage it at more advanced stages. Frequently cervical caries affects people over thirty years of age but it is often observed in children.

There are several stages of cervical caries development in children and adults. They are: a demineralized spot lesion, superficial caries, median and deep stages of dental caries. Cervical caries can be accompanied by different disorders of the thyroid, diabetes mellitus in particular. That is why it is necessary to undergo endocrinological examination along with cervical caries management. This disease often provokes the onset of circular caries in children. It can be accompanied by baby bottle tooth decay.

Most commonly they are seen in mandibular molars, followed by premolars, canines and incisors. This order is reversed in the maxilla. The buccal and interproximal surfaces are more susceptible than the palatal or lingual surfaces.

Cervical caries can occur in the areas of abrasion, erosion, and abfraction, or as primary root caries and recurrent decay.

### **Question 2. Etiology of cervical caries.**

All etiological factors of cervical caries are divided into two parts: intraoral and extraoral.

#### **Intraoral:**

- -Xerostomia.
- -Low salivary buffer capacity.
- -Poor oral hygiene.
- -Periodontal disease and periodontal surgery.
- -Gingival recession.
- -Frequency of carbohydrate intake.

- -Unrestored and restored coronal and root caries.
- -Overdenture abutments and removable partial dentures.
- -Malocclusion.
- -Abfraction lesions.
- -Tipped teeth which make areas of teeth inaccessible for cleaning.

**Extraoral:**

- -Advanced age.
- -Medications that decrease the salivary flow.
- -Antipsychotics, sedatives, barbiturates, and antihistamines.
- -Diabetes, autoimmune disorders (e.g. Sjogren's syndrome).
- -Radiation therapy.
- -Gender-males are affected more than females.
- -Lower educational and socioeconomic levels.
- -Physical disability where patients have limited manual dexterity for cleaning of teeth.
- -Limited exposure to fluoridated water.
- -Consumption of alcohol or narcotics.

Next, consider the most significant of these factors.

Continuous multiplication of microorganisms which are present in dental plaque and dental calculus is the principal cause of cervical caries development. The layer of tooth enamel is thinner in cervical regions and it is more difficult to brush the bacteria away from these zones. Hence, dental plaque accumulates very quickly. Some patients are predisposed to gingival layering which can result in cervical caries. Food gets into periodontal pockets and a great amount of pathogenic bacteria accumulate in these regions. These bacteria produce lactic acid which demineralizes both the enamel and the crown. Thus, the main causes of cervical caries are insufficient hygiene care and anatomic features of the patient's oral cavity.

In recent years, increased attention has been placed on the role of carbonated beverages, sports drinks, and their high sugar content in their combined chemical erosive effect on dentin. Patients who drink 4 to 6 bottles of a carbonated beverage per day, combined with poor oral hygiene, have a high risk of cervical carious lesions.

Besides that, adolescents undergoing orthodontic treatment are at risk for cervical carious lesions.

Xerostomia is also etiologic factor of cervical caries. The presence of xerostomia has been on the increase. Combined with gingival recession and exposed root surfaces, with xerostomia, teeth are at a greater risk of Class V carious lesions. Currently more than 400 medications can cause dry mouth. These medications include antihypertensives, antidepressants, analgesics, tranquilizers, diuretics, and antihistamines. Patients undergoing cancer therapy are susceptible to xerostomia. Chemotherapeutic medications can

affect both the flow and composition of saliva. Also, head and neck radiation can temporarily or permanently damage the salivary glands. After radiation, the protective ability of the saliva is also impacted by a decrease in the immunoglobulin in the saliva.

Other conditions can also cause a decrease in salivary flow. Patients with endocrine disorders, depression, anxiety and stress, and nutritional deficiencies may exhibit symptoms of dry mouth. Sjorgren's syndrome, an autoimmune disease, causes both dry mouth and dry eyes. Trauma to the head and neck area due to accidents or surgery can cause nerve damage that affects salivary flow.

### **Question 3. Symptoms of cervical caries.**

Cervical caries occurs on the gingival line. Classical symptoms like hypersensitivity to hot, cold and sweet are not observed at the initial stages of the disease progression. The signs of the disease can be noticed in external changes: that is the enamel darkens and white spots emerge. When the disease progresses painful reactions to temperature drops as well as to sour and sweet products may become frequent. On more advanced stages even tooth exposure to cold air may cause intolerable pain. Painful feelings while biting and chewing can be experienced. This disease is dangerous because of its rapid progression to the root of the tooth. Moreover, the affected tooth acquires a bad esthetic appearance. The disease may cause such complications as pulpitis, gingivitis and periodontitis. Cervical caries is transmitted from one tooth to another that is why treatment is obligatory. Moreover, advanced stages of cervical caries are accompanied by parulides, abscesses and phlegmon occurrence. These complications are life-threatening and the patient needs immediate hospitalization.

### **Question 4. Diagnosis of cervical caries.**

To diagnose cervical caries first of all doctor have to perform clinical examination is the. Tooth surface should be cleaned before examination since plaque covering the lesion can lead to misdiagnosis.

Often **patient's complaints** are short-term pain from hot, cold and sweet, presence of carious cavity, discomfort during teeth brushing, esthetic defect and others.

#### **Objective clinical examination. Visual-tactile method of diagnosis.**

Visual examination for diagnosing dental caries is a very popular method. It is based on the criteria such as cavitation, surface roughness, opacification and discoloration of clean and dried teeth under adequate light source.

*Advantage:*

- Preferred over probing because of harmful effects of probing.

*Disadvantages:*

- -Visual examination by a skilled clinician, in some cases, can be successful, but oftentimes, a large percentage of the occurrence of decay is too small to generate a distinctive visual signature for proper detection of caries even in advanced stages.
- -Discoloration which is found in normal healthy teeth, can be mistaken for the presence of caries.
- It is difficult to assess the level of caries penetration.

Tactile examination is the diagnostic method when the examiner detects softened tooth structure with dental probe. Since demineralization is a process that does not always involve sufficient softening of the enamel to be detectable by an explorer. When an explorer sticks, it's usually a good indication that there is decay beneath; however, when it does not stick, it does not necessarily mean that decay is not present. During the past 10 years the role of probing in caries detection has become a controversial issue. Sensitivity of these method is 62%, specificity – 84%.

*Disadvantages of probing:*

- It can produce traumatic defects in lesions arrested by plaque control alone.
- Does not improve accuracy of diagnosis.
- Inter operative variables.

During visual-tactile examination at the tooth cervical region we can see irregular or round or oval in shape lesion (spot or cavity) which often spread radially. The type of lesion depends on stage and disease activity.

**Initial root caries:**

- -White at first then may become light brown to yellow.
- Shallow, spreads laterally.
- Without patient symptoms.
- Hard on probing with moderate pressure.

**Active, progressing root caries:**

- Yellowish, light brown.
- Its covered by visible plaque.
- Soft or leathery on probing with light pressure.
- Its primarily detected by the presence of softness and cavitation.

**Inactive root caries:**

- Well-defined.
- Dark brownish or black in color.
- May be rough or smooth shiny surface but its cleanable.
- Usually not covered with plaque.
- Its seen in patients (usually older) whose oral hygiene and diet in recent years are good. Where discoloration of such areas is common and usually is associated with remineralization.



Despite the subjectivity inherent in interpreting the clinical signs of cervical caries diagnosis, good to excellent inter-examiner reliability has been reported in clinical studies. Clinical disagreement among different examiners can be attributed to several factors. Variation in an examiner's visual acuity (for example, presbyopia, color blindness) can obviously affect the interpretation of the presence or absence of cavitation and/or a color change on the root surface. Even more critical, however, is that there is frequently disagreement among examiners concerning the relative softness or hardness of the area examined due to differences in interpreting tactile sensitivity. There are no in vivo studies reported in the literature that compare clinical diagnosis with a histological assessment of the lesion. Although this is disappointing, it is not surprising because of the difficulty of conducting studies where teeth are removed and histologically examined following clinical examination.

**Diagnostic tests for cervical caries.** Clinicians look to diagnostic tests in the hope that they will be more reliable than clinical diagnosis and, therefore, can be used to replace clinical diagnosis. Selecting the most appropriate diagnostic test is therefore a complex matter that must take into account test characteristics, prevalence of the disorder, and the purpose of applying the test. For screening purposes, a highly sensitive test is generally preferred so that the number of false negative test results when the disorder is actually present is minimized. To assist with diagnosis, a highly specific test is preferred so that there will be few false positive test readings in the absence of disease. Test sensitivity and specificity, however, are uncalibrated measures of test performance.

Various tests have been used for the diagnosis of cervical caries. For the proximal surfaces, radiography produces good results, but the supporting evidence is weak. For cervical caries diagnosing we can use conventional or digital or bitewing radiography. The last are performed when carious cavity is localized at the tooth contact surfaces.

Accurate radiographs can help in diagnosis but they should be free from overlapping or burnout. Radiography taken on one occasion is unable to distinguish an actively progressing from a passive lesion, or a cavitated from a noncavitated surface. Deep dentinal lesions that are visible on a radiograph are more likely to be cavitated. Demineralized, noncavitated lesions may be arrested, but the main body of the demineralized, dentin usually remains radiolucent.

A considerable loss of mineral content is mandatory before lesion becomes visible on radiograph. The actual depth of lesion is always deeper than on radiograph.

*Grading:*

Grade I – Incipient,

II – Shallow, less than 0.5 mm,

III – Deep, pulpally involved.

Limitations of radiographs:

- Difficult to diagnose between residual and secondary caries.
- Cannot be visualized unless it reaches an additional stage.
- Cannot differentiate between activity of lesion.
- Marginal failure to be distinguished from secondary caries.

**Dye penetration method.** Special dyes can be useful for detecting root caries, these dyes stain the infected dentine and thus allow the clinician to detect caries.

There are two layers of decalcification can be identified in carious dentin: one layer which is soft and cannot be remineralized, a second layer, which is hard with intermediate calcification and can be remineralized. It is now clearly established that these dyes do not stain bacteria but instead stain the organic matrix of less mineralized dentin. This make them less specific because dyes do not stain bacteria nor delineate (trace an outline) the bacterial front but stain collagen associated with less mineralized organic matrix. Use of basic fuchsin in propylene glycol for the diagnosis and treatment of carious dentin has been given by Fusayama, 1980. This dye was found to be carcinogenic. To overcome this disadvantage, methylene blue was used, but methylene blue is slightly toxic.

*Dye-penetration methods using nowadays*

**a) For enamel caries:**

- Calcein: Complexes with calcium
- Fluorescent Dye: a) Brilliant blue b) ZygtoZX – 22.

Disadvantage is irreversible as dye reacts with nitrogen and hydroxyl groups of enamel.

**b) For dentin caries:**

- 0.5% basic fuchsin in propylene glycol,
- 1% acid red in propylene glycol.

It is known that the microorganisms most commonly associated with cervical caries are *Actinomyces viscosus/naeslundii*, *Streptococcus mutans*, *Lactobacilli*, and *Candida* species. That is why the tests determining the presence or absence of mutans streptococci and *Lactobacilli* are the known to be clinically helpful in diagnosing of cervical caries.

Fluorogenic enzyme assay estimates bacterial counts, particularly mutans streptococci and *Lactobacilli*, in plaque overlying root caries and, therefore, supports the evidence for mutans streptococci and *Lactobacilli* diagnostic tests.

Due to cervical caries is often connected with xerostomia for diagnosing we can also use such laboratory tests as salivary secretion rate test and salivary buffer effect test.

### **Question 5. Treatment of cervical caries.**

Treatment plan for cervical caries depends on the following factors:

- Clinical examination.
- Size of the lesion.
- Type, extent, and site of the lesion.
- Esthetic requirements.
- Physical and mental condition of the patient.

Root caries lesions are difficult to restore because of their location, which is near gingival margin or subgingival. For proper restoration, sufficient access and isolation are needed.

Proper access and isolation to treat root caries are very important, and ideally involve use of a rubber dam if the lesion is supragingival. To begin with it, root surface is cleaned to remove the plaque.

Then the excavation of carious tooth tissue is done and restoration walls are prepared. The margins and retention design depends on the restorative material used. For example:

- ✓ When a tooth is to be restored with amalgam, retention grooves are required occlusally and gingivally.
- ✓ For composites, beveling of the coronal margins of the preparation is required.
- ✓ If the location of the lesion is near the gingival margin or is subgingival cotton rolls and retraction cords can be used.
- ✓ If the lesion extends subgingivally and cannot be completely observed, even with the use of a retraction cord, a releasing incision may be required for completing the restorative procedure (Periodontal surgery).

There is a protocol for treatment of cervical caries that had putted by Billings in 1985 called (Index of Billings for root caries severity treatment) as following (Pic.):

- I. Grade 1: Incipient; no surface defect; need remineralizing therapy.
- II. Grade 2: Shallow; surface defect  $<0.5\text{mm}$ ; need recontouring.
- III. Grade 3: Cavitation; surface defect  $>0.5\text{mm}$ ; need filling.
- IV. Grade 4: Pulpal carious pulp exposure; need endodontic treatment + filling.

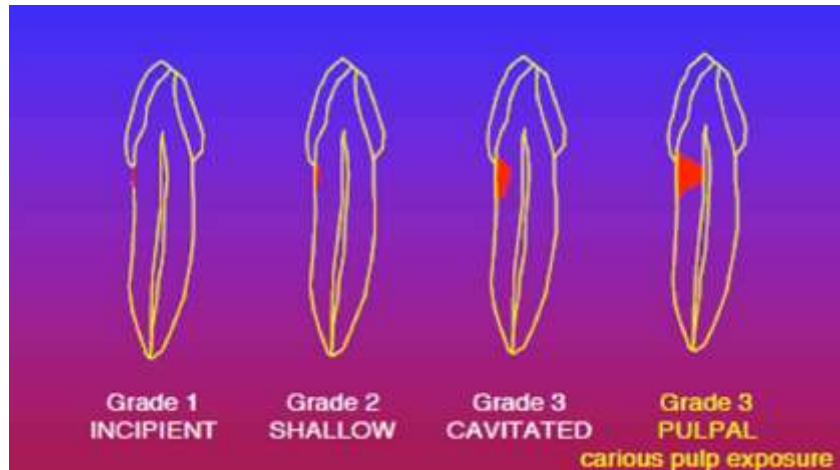


Figure 52. Index of Billings for root caries severity treatment.

There are different types of filling materials using for cervical caries treatment.

**Amalgam.** Properties:

- Easy to manipulate.
- Can be used in areas which are difficult to isolate.
- The margins are self-sealing.
- Lacks aesthetic appearance.
- No therapeutic effect.
- Cannot chemically bond to tooth structure.
- It requires the cutting of healthy tooth structure adjacent to the carious tissue for adequate retention of the restoration.

**Traditional glass-ionomers.** Properties:

- Biocompatible.
- It has chemical bond to tooth structure.
- Releasing fluoride over extended periods of time.
- Poor aesthetics.
- Excessive wear with time.

**Resin-modified glass-ionomers.** Properties:

- Biocompatible.
- Bond to tooth.
- Have thermal expansion and contraction characteristics that match tooth structure.
- Fluoride releasing feature; also it can be recharged by uptake of fluoride ions from the oral environment.
- They are aesthetic.
- Less brittle than the traditional glass ionomer.

**Resin composites.** Properties:

- Highly aesthetic materials.
- It bond to enamel and dentin.

- Hybrid composites possess improved strength and improved aesthetics compared with traditional resin composites.
- Microfilled composites are recommended for root surface restorations as they have lower elastic modulus than hybrid composites.
- Don't have any anti-cariogenic effect.

Resin composites are technique-sensitive materials and require proper isolation for the clinical success of the restoration.

Polymerization shrinkage associated with the curing of resin composites is another concern, since this can result in discoloration of the resin around the margins and in microleakage that leads to tooth sensitivity and secondary caries.

One of the most frequent clinical problems associated with class-II and class-V cavities in adhesive resin restorations is the weak link of restorative material to root dental structures, when the cervical margin is located below the cementum-enamel junction. In terms of cementum, the tissue-bonding properties have not been adequately elucidated. • It is well known that root surfaces exposed for a long period to the oral environment develop a superficial hypermineralized layer with limited permeability, compared with intact cementum. These surfaces may interfere in the marginal quality of root restorations, especially in elderly population. Very limited information exists on cementum-bonded restorations. Ferrari et al. in 1997 reported that cementum treated with dentine bonding systems is infiltrated by the resin, but the predictability of the bond is unclear.

Furthermore, it is still unclear (whether or not) the problem of bonding to cementum is related to the structure and properties of the tissue or to a limited effectiveness of the adhesive materials at the region.

However, the morphology of the periodontitis-affected cementum surface was highly variable, with islands of dense granular material. Based on these findings, mechanical removal of the superficial layer of the exposed cementum prior to any periodontal regenerative treatment has been advised. This treatment mode may be applied to improve adhesive bonding as well.

Modification of intact cementum surfaces to improve adhesion may include an eproteination step, prior to any adhesive treatment, in order to remove the high organic content and expose the inorganic substrate, like conditioning with aqueous solutions of sodium hypochlorite (NaOCl).

Sandwich technique is another solution to solve adhesion of composite to root surface.

#### **Fluoride-containing resin composites. Properties:**

- Fluoride-containing resin composites release only small amounts of fluoride.
- It has little ability to recharge from the oral environment.

- Therefore, they are not recommended for use with high caries-risk patients, but can be used where aesthetics is a concern.

**Compomers.** Properties:

- They are polyacid-modified resin composites.
- They have possess properties of both glass ionomer and resin composites.
- They leach fluoride, but to a lesser extent than glass ionomer.
- They bond to both enamel and dentin.
- They can be used in low-stress areas where esthetics is a concern.

**Table 38.Characteristics of different filling materials for cervical caries treatment**

<i>Material</i>	<i>Flouride release</i>	<i>Adhesive</i>	<i>Aesthetics</i>
Amalgam	None	No	Low
Glass ionomer	High	Yes	Moderate
Resin- modified glass ionomer	High	Yes	Moderately.High
Resin composite	None	Yes, with bonding	High
Flouride-containing resin composite	Little	Yes, with bonding	High
Compomer	Moderate	Yes, with bonding	Moderately. High

Treatment of root surface caries should be directed and customized to the individual case by classifying patients in risk groups to achieve maximum results.

The use of resin-modified glass ionomer materials is recommended for these restorations because of their anti-cariogenic properties in patients with a high caries risk.

**Question 6. Prevention of cervical caries.**

1. Proper dental hygiene should be maintained: high-quality tooth brush and tooth paste must be used to clean the teeth. To brush in between the teeth it is necessary to use dental floss. Gingival massage and mouth rinsing with herbal decoctions are recommended to promote enamel regeneration.

2. Well-balanced nutrition is a key factor of dental health. Dental prophylaxis also presupposes regular dental examinations, even when nothing bothers the patient. It is necessary to remember that sweets themselves do not cause caries. It occurs as a result of poor hygiene of the

oral cavity where the microorganisms multiply. That is why parents should monitor the process of their children's teeth brushing.

**3.** The dentist will perform professional teeth brushing, remove dental calculus, whiten and remineralize tooth enamel.

**4.** Special attention should be given to root caries-prone patients who are wearing dental prostheses. This can be done by proper management of soft tissues during fixed prosthesis procedures and avoiding the placement of restoration margins apical to the surrounding tissue to avoid plaque accumulation.

**5.** In patients with low salivary flow, xylitol-containing chewing gum which stimulates salivary flow and decreases plaque formation has shown to decrease the caries.

**6.** The use of topical fluoride should be advocated because it promotes the remineralization process and reduces the rate of demineralization. There are numerous methods by which fluoride can be supplied:

- ✓ Exposure to fluoride in drinking water results in increasing resistance to root caries.
- ✓ Topical fluoride products are available as 0.05% sodium fluoride rinse, 0.12% chlorhexidine rinse, and as 1.1% neutral sodium fluoride gel in a 5 minutes tray technique, with 4 applications over 2-4 weeks.
- ✓ Other products are dentifrices containing 1100 ppm sodium fluoride.
- ✓ Fluoride chewing gum which is effective especially in patients with low salivary flow.
- ✓ Fluoride-containing varnishes have also been effective against root caries.

### **Tests to the topic**

**1. Cervical caries is often accompanied by:**

- a. Kidney diseases.
- b. Liver diseases.
- c. Endocrine diseases.
- d. All of the above.

**2. Most commonly cervical caries are seen on the:**

- a. Incisors.
- b. Canines.
- c. Mandibular molars and premolars.
- d. All of the above.

**3. What surfaces are more susceptible to the cervical caries?**

- a. Palatal or lingual.
- b. Buccal and interproximal.
- c. All of the above.

**4. What etiological factors of cervical caries are intraoral?**

- a. Xerostomia.
- b. Low salivary buffer capacity.
- c. Poor oral hygiene.
- d. Periodontal disease and periodontal surgery.
- e. Gingival recession.
- f. All of the above.

**5. What etiological factors of cervical caries are extraoral?**

- a. Medications that decrease the salivary flow.
- b. Antipsychotics, sedatives, barbiturates, and antihistamines.
- c. Diabetes, autoimmune disorders (e.g. Sjogren's syndrome).
- d. Radiation therapy.
- e. All of the above.

**6. Patient's with cervical caries complain on:**

- a. Prolonged ache from hot, cold and sweet.
- b. Short-term pain from hot, cold and sweet.
- c. All of the above.

**7. What are the types of cervical carious lesions:**

- a. Initial root caries.
- b. Active, progressing root caries.
- c. Inactive root caries.
- d. All of the above.

**8. Treatment plan for cervical caries depends on the following factors:**

- a. Clinical examination.
- b. Size of the lesion.
- c. Type, extent, and site of the lesion.
- d. Esthetic requirements.
- e. All of the above

**9. What is the protocol for the treatment of cervical caries on the initial stage?**

- a. Remineralizing therapy.
- b. Recontouring.
- c. Filling.
- d. Endodontic treatment + filling.
- e. Prosthetics.



**10. What are the types of filling materials using for cervical caries treatment?**

- a. Amalgam.
- b. Traditional glass-ionomers.
- c. Resin-modified glass-ionomers.
- d. Resin composites.
- e. Compomers.
- f. All of the above.

## **LESSON 15. ERRORS AND COMPLICATIONS IN THE DIAGNOSIS AND TREATMENT OF DENTAL CARIES**

The questions to be studied for the learning of the topic:

1. Errors and complications arising during carious cavity preparation.
2. Errors and complications arising during carious cavity filling.
3. Errors and complications arising after dental caries treatment.
4. Preventive measures in dental caries treatment.

### **Question 1. Errors and complications arising during carious cavity preparation**

During treatment of dental caries, doctor performs a variety of manipulations, not very thorough or improper performance of which can lead to some kind of complications. These errors can occur both during the actual surgical treatment, preparation of carious cavities and on the stages of carious cavity filling and at a different times after sealing. It is therefore advisable to divide them into complications arising during the preparation of carious cavities and during filling of carious cavity, and the complications that arise after treatment of caries.

Knowledge of possible mistakes and errors during carious cavity preparation and filling material placement will prevent young clinician from the complications arising as the result of mistakes.

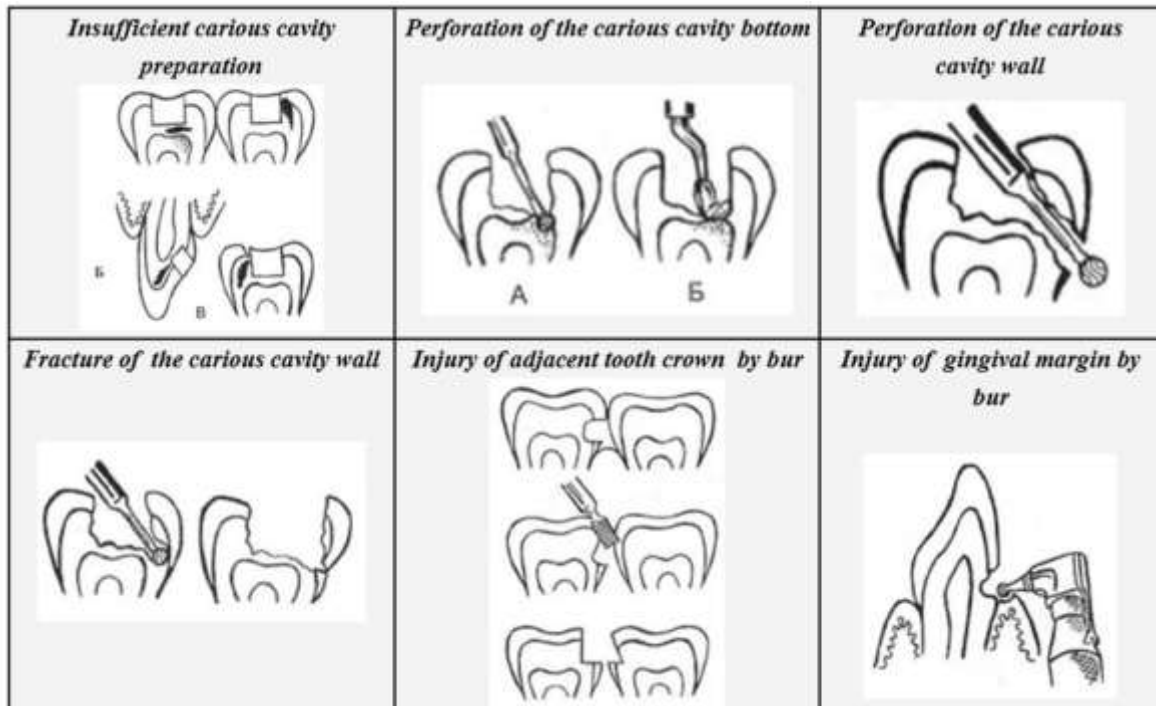
#### ***Errors and complications arising during carious cavity preparation***

1. Insufficient carious cavity preparation may lead to secondary caries, thus progressing of caries process and possible development of pulpitis or filling loss.

2. Perforation of the carious cavity bottom or carious cavity wall and fracture of the carious cavity wall may happen due to not proper fixed hand of clinician thus leading to such complications. Perforation of carious cavity floor may happen in the case of acute deep dental caries, when bottom is softened and thin layer of demineralised dentine separates carious cavity from tooth cavity.

4. Injury of adjacent tooth crown by bur may happen when visible control of operative field is not provided.

5. Injury of gingival margin by bur may happen during preparation of carious cavities that goes deep under the gums or good vision of operative field was not provided.



*Figure 53. Errors and complications arising during carious cavity preparation*

## **Question 2. Errors and complications arising during carious cavity filling.**

1. Absence of a contact point, hanging edges of a filling and placement of a single filling in adjacent carious cavities will lead to inflammation of intradental papilla, thus causing pain to the patient and development of periodontal diseases. That's why during restoration of II class by Black (proximal cavities) it is necessary to use matrix holder and matrices in order to restore contact point, thus preventing these complications.

2. Formation of high occlusion usually happen when filling is not adjusted to the bite, when high spots are left, this will lead to development of apical periodontitis in future, such tooth will change its color to grey shades and will be painful while biting (diagnostic feature).

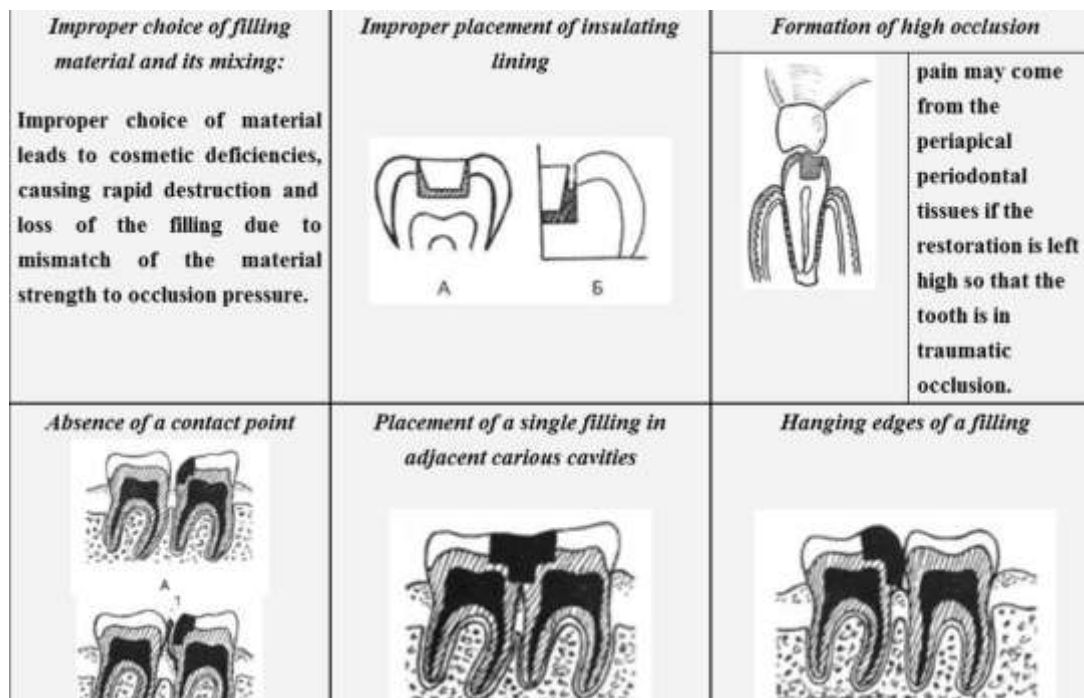


Figure 54. Errors and complications arising during carious cavity filling

### Question 3. Errors and complications arising after dental caries treatment.

1. Inflammation (necrosis) of the pulp.
2. Secondary caries.
3. Papilitis (inflammation of an intradental papila).
4. Acute or chronic course of an apical periodontitis.
5. Colour change of the tooth crown.
6. Displacement, fracture and loss of filling.
7. Inadequate colour of filling to the colour of tooth enamel.

### Question 4. Preventive measures in dental caries treatment.

*Preventive measures at the stage of cavity preparation.* In preservation dentistry, the preparation of dental hard tissues is based on the removal of carious lesions in dental tissue and on the ensuring of retention of the filling with regard to the prevention of dental caries. Residues of a carious lesion left on the cavity walls and the pulp wall result in secondary or recurrent caries formation with its consequences.

The identification of recurrent caries, particularly in the fillings with a good marginal closure is a diagnostic problem which cannot sometimes be solved even by using dental radiography.

The main aim of dental caries treatment is to preserve healthy dental pulp. The current choice of preparation tools considering effectiveness and the used filling material is a basis for the prevention of pain sensation during the preparation procedure.

When manual preparation instruments are used, the patient feels the instrument's pressure. If low-speed rotating preparation tools are used, painful sensations are distributed evenly between pressure, temperature and vibration sensations. With high-speed rotating tools, the sensation of pain due to pressure and vibrations is decreased but temperature irritation increases. With ultra-high-speed instruments, 80 – 90% of energy is converted into heat.

**Table 39. The effect of the instrument's speed on pain sensation**

<i>Manual preparation</i>	<i>Pressure</i>	<i>Sensation</i>
Low speed	Pressure	///
	Vibrations	///
	Heat	///
High speed	Pressure	/
Ultra-high speed	Vibrations	/
	Heat	////////

As a result, when the prepared area of the tooth is not cooled properly, thermal damage to odontoblasts may occur together with irreversible changes in the pulp.

Insufficient cooling may occur when the flow of water from the jet does not wash the prepared field due to the shape of the cavity and the angle of a preparation tool.

Unpleasant to painful sensation will arise due to the vibrations of rotating tools which are not centred properly or have been used for a longer period of time. Pain increases particularly in patients with the damaged periodontium.

Pain relief and reduction in the preparation time depend on the effectiveness of the preparation tools used (i.e. on the type, quality and the way of using the tools). When sharp tools are used, approximately one half of kinetic energy delivered to the axis of a rotating tool during drilling and polishing will change into heat. This portion increases with blunt instruments; almost all energy will change into heat when totally blunt tools are used. The effectiveness of drilling and polishing strongly depends on the cross-section of a chip and the cutting speed whose magnitude depends on the tool's diameter and the number of revolutions. An increase in the cutting speed at an ultra-high number of revolutions enables one to make a chip of a smaller diameter, with lower pressure applied on the tool and thus lesser pain during treatment. Reduction in the number of revolutions during preparation depends on the pressure on the tool. The most significant reduction in the number of revolutions is in turbine handpieces with a direct drive where pressure causes the number of revolution to decrease by 25 - 30%. Improper

and damaged rotating components result in vibrations and the overheating of the handpiece. This also increases the tool's noise level.

Lege artis treatment is crucial when dental caries is found.

The cavity preparation depends on the localization of dental caries, the extent of the loss of dental hard tissue and the filling material used. Conventional preparation procedures follow basic principles.

The main principle for dental caries treatment is to remove the matter changed by carious lesion. Complications usually occur during the treatment of caries located near the pulp when the dentist failed to remove the carious dentin or when the removal of carious matter was too radical irrespective of the anatomical size of the pulp cavity, increasing the risk of perforating the pulp cavity.

When the principles of preventive extension during cavity preparation is maintained, this will ensure the extension of cavity up to the point of self-cleaning. Microbial plaque accumulates at a higher extent on irregularly positioned teeth. Treatment of dental caries for irregularly positioned teeth depends not only on the extent of loss of dental tissues, but also on a degree of seriousness of a particular irregularity. In a large number of cases, the performance of a conventional treatment is not possible and it is necessary to apply materials with adhesive bonds. At the extensive loss of hard dental tissue, there is a risk of subsequent fracture of a crown. In such cases it is therefore necessary to do prosthetic treatment or extract the abnormally positioned or markedly destructed tooth.

Through their attachment to dental hard tissues (mechanical, chemical), composite materials are able to reduce preparation procedures and save dental hard tissues (preventive fillings), achieving the best possible aesthetic effect at the same time.

The prepared cavity must ensure the sufficient retention of a filling. The preparation depends on the type of the material used (amalgam filling, cast fillings, inlays made of different materials, composite filling materials and glass ionomer cements). Non-compliance with retention principles for individual materials will result either in the release of a filling or failure to achieve its mounting. Fillings are exposed to chewing pressure. The respective kind of the material also specifies the need of sufficient depth of preparation to ensure the resistance of individual filling materials. Shallow prepared cavities fail to provide sufficient firmness for the material and will break upon biting. Damage to the filling may also occur in high fillings during the adjustment of occlusion/articulation relations. The risk of breaking the filling is highest in MOD cavities and in Class IV cavities with the insufficient adjustment of articulation relations. The broken filling may irritate the marginal periodontium causing acute papillitis or periodontal abscess.

The strength of composite materials stems from mechanical binding to the bevelled and etched enamel, and increases with chemical binding to the enamel and dentin depending on the used binding system. When composite filling materials are used, mechanical and chemical binding shortens the preparation procedure. The depth of the prepared cavity is not governed only by the principle of the filling resistance. The extent of carious lesion and the size of pulp cavity (age factor, anatomical relationship of individual teeth) are crucial factors. When the caries is located near the pulp, it is necessary to prepare in the close vicinity of the top wall of the pulp cavity. Due to the risk of perforation, manual tools are required for performing gentle preparation and completing the preparation.

When the preparation of the cavity is carried out, it is necessary to ensure not only the sufficient stability of the filling against chewing pressure but also the proper resistance of the remaining dental hard tissues. With regard to the resistance of dental hard tissues, it is necessary to comply with the “cusp rule” during the preparation. The weakened cusps fail to provide sufficient support to the remaining dental tissue and the crown or root may fracture upon biting. The fractures expanding below the dentogingival junction and longitudinal fractures of the root are frequently an indication for the subsequent extraction of a tooth. It is therefore necessary to consider the indications for the use of plastic or cast dental fillings. In the case of a large loss of dental hard tissue, it is necessary to apply prosthetic treatment.

The extent of the preparation can be reduced in the case of small caries on the occlusion surfaces of molars with huge cusps. The use of molar composite materials is recommended.

In order to prevent secondary caries, the enamel margin of the cavity, where the amalgam filling will be placed, must be treated properly. The enamel must be underlain with dentin. The use of manual tools is recommended for the adjustment of the enamel.

The main aim of using composite materials is to ensure the greatest possible mechanical retention of a composite. The insufficient angle of the enamel or errors in subsequent phases of the procedure will lead to the failure to close the cavity, resulting in secondary caries.

When caries treatment uses cast dental fillings, the bevelling of enamel's edges is the main condition to ensure the cavity's proper closure.

#### ***Preventive measures at the stage of cavity filling***

Secondary caries or poor-quality fillings can also occur as a result of the working procedure used to fill the cavity with the filling material.

Among the most common mistakes associated with amalgam fillings is the incorrect selection of matrix and its insufficient fixation in Class II cavities. The injured dentogingival junction is painful and gingival bleeding

will occur. Failure to ensure the dry working field makes the lege artis completion of the definitive filling impossible.

The cavity has to be treated using a temporary filling and the definitive filling has to be made at a next visit.

Damage to the marginal gingiva will also occur at the improper choice and attachment of sealing wedges in the interdental space. If the matrix is sealed insufficiently, amalgam during condensation will penetrate across the cavity's margin into the interdental space. The removal of non-solidified amalgam from the interdental space using manual tools is insufficient and will always result in injury to the marginal gingiva. It is therefore necessary to make a new filling. Solidified overhanging fillings (of any material) will irritate the marginal gingiva and cause acute and chronic pathological conditions of the periodontium.

When the cavity is being filled, it is necessary to apply the amalgam into the cavity in small portions and allow it to condensate properly. Insufficient condensation is manifested by uneven filling. The risk is greatest at the filling of the gingival ledge in Class II cavities. Such fillings may break upon bite and irritate the marginal periodontium (pain upon occlusion).

One important part of the working stage in the case of cast fillings is that the cast in the cavity will be tested. Thorough examination of the marginal closure of the cavity is the main prerequisite for a proper closure. The presence of a fissure between the filling and the enamel will always necessitate the formation of a new model.

In the case of composite materials, it is necessary to comply with the working procedure for cavity filling. Shrinkage during polymerization arising due to the solidification of materials defines the suitability of using different forms of composite filling materials. Large cavities and destructed crowns are treated using photopolymerizing materials. The quality of the filling made of photopolymerizing composite material is also affected by the quality of the lamp used, the type of material and the binding system used. Failure to comply with the working procedure at each stage of the construction of a filling will result in the poor-quality filling.

#### ***Preventive measures at the stage of the final treatment of fillings***

The quality of the filling also depends on the filling's final treatment. The correction of occlusion and articulation relations will prevent the elevated bite onto the filling and the overloading of the periodontium. The insufficiently adjusted height of the filling will result in the broken filling and its release from the cavity. High fillings after solidification may cause acute or chronic damage to the periodontium.

With regards to the prevention of secondary caries, the treatment of the filling's surface is important. Amalgam fillings as well as cast dental fillings must be polished thoroughly, particularly at the filling's margins.



There is currently a wide range of different polishing systems ensuring the perfect polishing of all kinds of fillings. The polishing of the solidified filling's surface is also important in composite fillings. After the adjustment of occlusion and articulation, the thorough polishing of the filling's surface is a prerequisite for good aesthetic appearance. The polished surface of the filling will also ensure less accumulation of microbial plaque.

### **Tests to the topic**

#### **1. Errors and complications arising during carious cavity preparation are**

- a. Insufficient carious cavity preparation.
- b. Perforation of the carious cavity bottom or carious cavity wall.
- c. Injury of adjacent tooth crown.
- d. Injury of gingival margin.
- e. All answers are right.

#### **2. Errors and complications arising during carious cavity filling are**

- a. Injury of gingival margin.
- b. Absence of a contact point.
- c. Formation of high occlusion.
- d. Injury of adjacent tooth crown.
- e. All answers are right.

#### **3. Errors and complications arising after dental caries treatment are**

- a. Inflammation (necrosis) of the pulp.
- b. Secondary caries.
- c. Papilitis (inflammation of an intradental papilla).
- d. Acute or chronic course of an apical periodontitis.
- e. Colour change of the tooth crown.
- f. Displacement, fracture and loss of filling.
- g. All answers are right.

#### **4. What are the main types of errors and complications during caries treatment?**

- a. Errors and complications arising during carious cavity preparation.
- b. Errors and complications arising during carious cavity filling.
- c. Errors and complications arising after dental caries treatment.
- d. All answers are right.

#### **5. Insufficient carious cavity preparation may lead to:**

- a. To secondary caries.
- b. Development of pulpitis.

- c. Filling loss.
- d. All answers are right.

**6. Injury of gingival margin by bur may happen when:**

- a. Carious cavity goes deep under the gums.
- b. Good vision of operative field was not provided.
- c. Contact point is absent.
- d. All answers are right.

**7. In what cavities risk of filling breaking is highest?**

- a. In mod cavities.
- b. In class iv cavities.
- c. In class i cavities.
- d. All answers are right.

**8. The most common errors associated with amalgam fillings are:**

- a. Incorrect selection of matrix.
- b. Insufficient fixation in class ii cavities.
- c. Secondary caries.
- d. All answers are right.

**9. The quality of the filling made of photopolymerizing composite material depends on:**

- a. The quality of the lamp used.
- b. The type of material.
- c. The binding system used.
- d. All answers are right.

**10. Pulp necrosis occurs when it is heated to:**

- a.  $45^{\circ}$ .
- b.  $55^{\circ}$ .
- c.  $70^{\circ}$ .

## LESSON 16. POSSIBILITIES OF FILLING MATERIAL CHOICE IN DIFFERENT CASES

The questions to be studied for the learning of the topic:

1. Classifications and properties of restorative materials.
2. Direct composite restorative materials. Composite resins—constituents and properties.
3. Composite resins - practical points. Polymerization of Composite
4. Glass-ionomer cements (GICs) - classification, constituents and properties.
5. Basic Concepts of Adhesion
6. Advantages and disadvantages of different types of restorative materials.

### **Question 1. Classifications and properties of restorative materials.**

Dental caries is a major public health problem globally. Despite much effort in health promotion and disease prevention, dental restorations are still needed.

In the case of dental treatment, diseased tissue is removed and teeth restored with appropriate material(s).

The teeth themselves are composed of enamel, dentine, and pulpal tissues each with quite different properties that must be taken into account when selecting a material with which to restore them.

#### ***Restorative materials for direct restorations***

- Composite resins
- Enamel and dentine bonding
- Amalgam
- Glass ionomers
- Resine-modified glass ionomer/composite-based
- Cements

#### ***Restorative materials for indirect restorations***

- Composite resins
- Amalgam
- Casting alloys
- Wrought alloys
- Dental ceramics
- CAD/CAM

### **Properties of dental materials**

#### ***Coefficient of thermal expansion***

The fractional  $\uparrow$  in length for each degree of temperature  $\uparrow$ .

**Creep.** The slow plastic deformation that occurs with the application of a static or dynamic force over time.

**Elastic modulus.** A measure of the rigidity of a material, defined by the ratio of stress to strain (below elastic limit).

**Fatigue.** When cyclic forces are applied, a crack may nucleate and grow by small increments each time the force is applied. In time the crack will grow to a length at which the force results in fracture through the remaining material.

**Hardness.** Resistance to penetration. A number of hardness scales are in use (e.g. Vickers, Rockwell). Between these scales hardness values are not interchangeable.

**Resilience.** The energy absorbed by a material undergoing elastic deformation up to its elastic limit.

**Stiffness.** An indication of how easy it is to bend a piece of material without causing permanent deformation. It is dependent upon the elastic modulus, size, and shape of the specimen.

**Strain.** Change in size of a material that occurs in response to a force. It is the change in length divided by the original length.

**Stress.** Internal force per unit cross-sectional area acting on the material. Can be classified according to the direction of the force: tensile (stretching), compressive, or shear.

**Thermal conductivity.** Ability of a material to transmit heat.

**Thermal diffusivity.** Rate at which temperature changes spread through a material.

**Toughness.** The amount of energy absorbed up to the point of fracture. A function of the resilience of the material and its ability to undergo plastic deformation rather than fracture.

**Wear.** The abrasion (mechanical or chemical) resistance of a substance.

**Wettability.** Ability of one material to flow across the surface of another, determined by the contact angle between the two materials and influenced by surface roughness and contamination. The contact angle is the angle between solid/liquid and liquid/air interfaces measured through the liquid.

**Yield strength (or elastic limit).** The stress beyond which a material is permanently deformed when a force is applied.

#### **Clinically important questions**

- ✓ Details of the chemical constituents.
- ✓ Handling characteristics, e.g. presentation, mixing, working time, setting time, and dimensional changes on setting.
- ✓ Performance in service.
- ✓ Cost.
- ✓ Shelf-life.
- ✓ Does the material meet the relevant ISO standard?

## Question 2. Direct composite restorative materials. Composite resins—constituents and properties.

A generalized definition of a composite is a multiphase material that exhibits the properties of both phases where the phases are complimentary, resulting in a material with enhanced properties

### Organic phase

Resin - Methyl methacrylate monomer

### Inorganic phase

Fillers - Glass particles

The modern composite resin is a mixture of resin and particulate filler, the handling characteristics of which are determined largely by the size of the filler particles and method of cure.

### Constituents

**Resin.** Most composite resins are based on either Bis-GMA (addition product of bisphenol A and glycidylmethacrylate) or urethane dimethacrylate plus adiluent monomer, triethyleneglycol dimethacrylate (TEGDMA).

The chemical structure of Bis-GMA, a resin invented by Ray Bowen. It also is referred to as Bowen's resin.

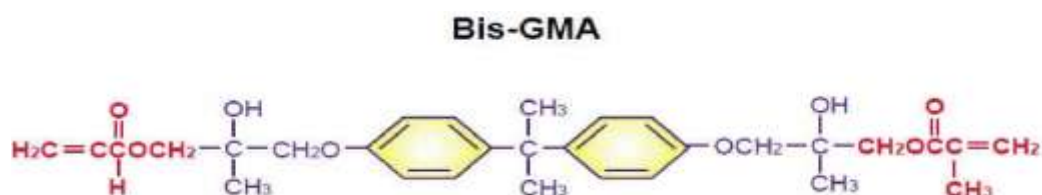


Figure 55. The chemical structure of triethyleneglycol dimethacrylate Bis-GMA

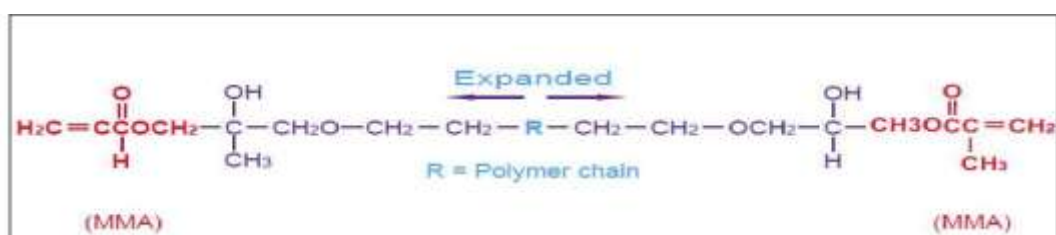


Figure 56. The chemical structure of triethyleneglycol dimethacrylate TEGDMA

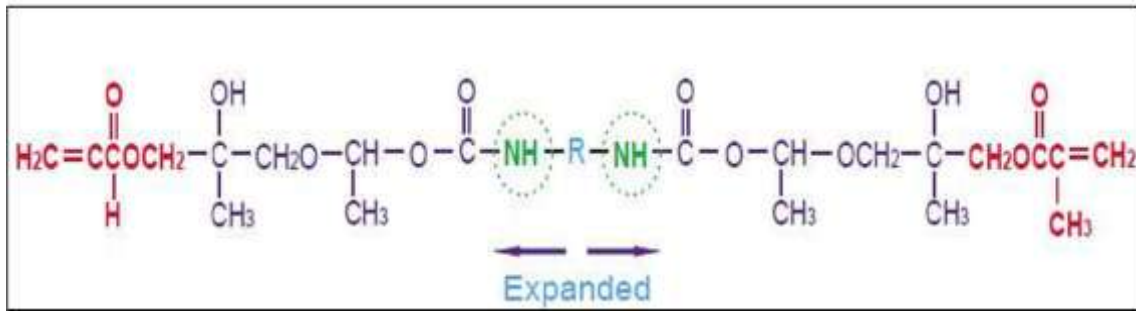


Figure 57. The chemical structure of difunctional urethane resins - Urethane dimethacrylate (UDMA)

·  $R$  = a number of carbon compounds that can be used to lengthen or alter the properties of the monomer.

· Nitrogen in the form of  $NH-R-NH$  is the urethane component

**Filler(e.g. quartz, fused silica, glasses such as aluminosilicate and borosilicate).**

- This is the reinforcing phase of direct dental restoratives
- This is based on glass or ceramic particles.
- Incorporation of these inorganic particles imparts improved strength and wear properties, decreased CTE, reduced polymerization shrinkage.
- Incorporation of heavy metals into the filler provides radiopacity.

Currently particles dimensions of fillers ranging from nm to mm.

***Confers the following benefits on the composite resin:***

- Compressive strength, abrasion resistance, modulus of elasticity, and toughness.
- Thermal expansion and setting contraction.
- Aesthetic qualities.

***Positive Properties of monomers (resin).*** Viscosity, Flowability

***Negative Properties of monomers (resin)***

- a. polymerization shrinkage
- b. Coefficient of Thermal Expansion (CTE) of resin composites and tooth structure. The CTE of tooth structure ranges from 9 to 11 ppm/\_C, compared with 28 to 50 ppm/\_C for dental composite restoratives
- c. Water absorbtion

**Composite restorative materials** consist of a continuous polymeric or resin matrix in which an inorganic filler is dispersed. Particles of filler are coated by silane coupling agent, which increases the strength of the composite but also reduces its solubility and water absorption. Inorganic filler phase significantly enhances the physical properties of the composite (compared with previous materials) by increasing the strength of the restorative material and reducing thermal expansion.

### **Composites classification**

Are classified primarily on the basis of the size, amount, and composition of the inorganic filler:

- ✓ macrofill composites
- ✓ microfill composites,
- ✓ hybrid composites (including traditional hybrid, microhybrid, and nanohybrid composites),
- ✓ nanofill

Are classified on the basis of their handling characteristics:

- ✓ Packable
- ✓ flowable

**Macrofill composites** were the first type of composites introduced in the early 1960s. Contained approximately 75% to 80% inorganic filler by weight.

The average particle size was approximately 8 mkm

Exhibit a rough surface texture

Are susceptible to discoloration from extrinsic staining

Have a higher amount of initial wear at occlusal contact areas than do microfill or hybrid types

Most macrofill composites currently have been supplanted by hybrid composites

**Microfill composites** were introduced in the late 1970s.

- ✓ Contain colloidal silica particles whose average diameter is 0.01 to 0.04 mkm.
- ✓ Have a smooth, polished surface.
- ✓ Have an inorganic filler content of approximately 35% to 60% by weight.
- ✓ Physical mechanical characteristics are inferior.
- ✓ Low modulus of elasticity - better protecting the bonding interface and allow microfill composite restorations to flex during tooth flexure.
- ✓ Are the material of choice for restoring Class V lesions.

**Hybrid Composites.** Were developed to combine physical and mechanical properties of macrofill composites with the typical properties of the microfill composites.

These materials have an inorganic filler content of ~ 75% to 85% by weight;

The filler has been a mixture of microfiller and small filler particles that results in smaller average size (0.4-1  $\mu\text{m}$ ) than of conventional composites.

Because of the relatively high content of inorganic physical and mechanical characteristics are superior to those of conventional composites.

**Nanohybrid composites.** Current versions of hybrid composites contain ultrasmall nanofillers resulting in superior characteristics.

**Nanofill composites** contain filler particles that are extremely small (0.005-0.01  $\mu\text{m}$ ) and agglomerated. High filler level proves good physical properties and improved esthetics. The small particle size makes nanofills highly polishable. Nanofill and nanohybrid composite are the most popular composite restorative materials in use. These composites have almost universal clinical application

**Packable composites** are designed to be more viscous, similar to that of amalgam. Because of increased viscosity and resistance to packing, some lateral displacement of the matrix band is possible.

Their development is an attempt to accomplish two goals:

- (1) easier restoration of a proximal contact
- (2) similarity to the handling properties of amalgam.

But because of the increased viscosity, *it is more difficult to attain optimal marginal adaptation, prompting some clinicians to first apply a small amount of flowable composite along proximal marginal areas to enhance adaptation.*

**Flowable composites** have lower filler content, lower wear resistance and lower strength compared with the more heavily filled composites.

They also exhibit much higher polymerization shrinkage.

Flowable composites to be more appropriate for use in

- a. as the first increment placed as a stress-breaking liner under posterior composites.
- b. as first small increments in the proximal box of a Class II restoration in an effort to improve marginal adaptation.
- c. some small Class I restorations
- d. as pit-and-fissure sealants
- e. as marginal repair materials.
- f. as bulk-fill materials to be used to restore posterior teeth - Bulk-fill posterior (3M ESPE).

But physical properties of flowable composites are generally poor, and the long-term performance of such restorations is not yet proven. They should never be placed in areas of high proximal or occlusal stress because of their comparatively poor wear resistance.

#### Advantages of composites

- ✓ Esthetic.
- ✓ Conservative in tooth structure removal (less extension, uniform depth not necessary, mechanical retention usually not necessary).
- ✓ Less complex when preparing the tooth.
- ✓ Insulating; having low thermal conductivity.
- ✓ Used almost universally.



- ✓ Bonded to tooth structure, resulting in good retention, relatively low microleakage, minimal interfacial staining, and increased strength of remaining tooth structure.
- ✓ Repairable.

#### Disadvantages of composites

The primary disadvantages of composite restorations relate to potential gap formation and procedural difficulties (technique). Composite restorations are more technique-sensitive because the operating site must be appropriately isolated and proper technique is obligatory in the placement of etchant, primer, and adhesive on the tooth structure (enamel and dentin).

1) Gap formation is a result of the forces of polymerization shrinkage (PS) of the composite material because of PS greater than the initial early bond strength of the material to dentin.

2) A gap can result from improper insertion of the composite by the clinician.

#### ***Procedural difficulties of composite restorations***

- Are time-consuming, because bonding usually requires multiple steps; insertion demands sequence of material portions;
- Establishing proximal contacts, axial contours, embrasures, and occlusal contacts demand certain techniques
- Finishing and polishing procedures have a few stages.

### **Question 3. Composite resins-practical points. Polymerization of Composite.**

Composite materials shrink while polymerizing (polymerization shrinkage). Important clinical techniques must be performed to avoid pulling away from the preparation walls during polymerization.

#### ***Polymerization methods***

##### Self-cured materials require mixing two components

- 1) the risk for air inclusion in the mixture and internal porosity is greater
- 2) working time to insert the self-cured material is restricted by the speed of chemical reaction
- 3) color stability is lower
- 4) the direction of PS is centralized (toward the center of the mass)

##### Light-cured method

- 1) Light-curing systems (LEDs)
- 2) Light-cured materials provide increased working time during insertion, greater color stability, less internal porosity
- 3) High-intensity and high-speed curing increase the stresses from heat generation and PS
- 4) Careful control of the amount and insertion point of the material
- 5) Appropriate use of an adhesive on the prepared tooth structure

**C-factor is effects of polymerization shrinkage** - C-factor is the ratio of bonded surfaces to the unbonded, or free, surfaces in a tooth preparation. The higher the C-factor, the greater is the potential for bond disruption from polymerization effects.

A Class IV restoration (one bonded surface and four unbonded surfaces) with a C-factor of 0.25 is at low risk for adverse polymerization shrinkage effects. A Class I restoration with a C-factor of 5 (five bonded surfaces, one unbonded surface) is at much higher risk of bond disruption associated with polymerization shrinkage, particularly along the pulpal floor.

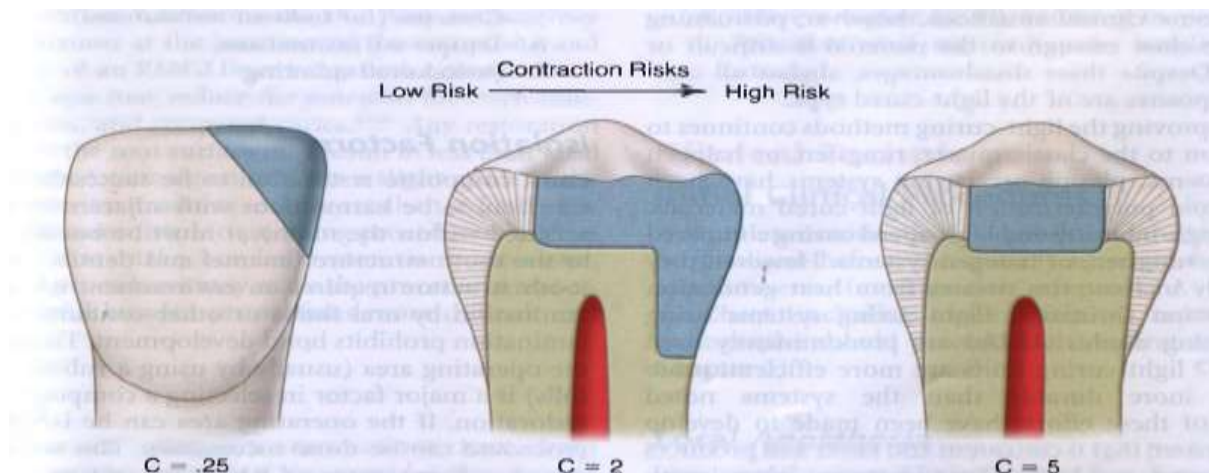


Figure 58. Reducing of polymerization shrinkage

*Internal stresses can be reduced by using*

- (1) “soft-start” polymerization instead of high-intensity light-curing;
- (2) incremental additions to reduce the effects of polymerization shrinkage;
- (3) a stress-breaking liner such as a filled dentinal adhesive, flowable composite, or RMGI.

Another approach is usage a different polymer as the matrix.

Typical hybrid composites using BIS-GMA or UDMA as the matrix shrink approximately 2.4% to 2.8%.

Microfilled and flowable composites shrink considerably more because they are less highly filled.

One product, Filtek LS (3M ESPE) uses a silorane polymer matrix and the linear shrinkage of this composite is approximately 0.7%.

**The method of polymerization of a composite may affect:**

- ✓ the technique of insertion,
- ✓ direction of polymerization shrinkage,
- ✓ finishing procedure,
- ✓ color stability,
- ✓ amount of internal porosity in the material.

#### **Question 4. Glass-ionomer cements (GICs) -classification, constituents and properties.**

The original GICs are water based materials which set by an acid-base reaction between a polyalkenoic acid and a fluoroaluminosilicate glass.

##### Setting reactions

- Powder and liquid are mixed.
- The acid etches the glass that results in a release of calcium, aluminium, sodium and fluoride ions into solution.

This is an acid-base reaction where the water serves as the medium for the reaction.

- The metal ions react with the carboxyl (COO) groups to form a polyacid salt, which becomes the cement matrix.
- The surface of the glass becomes a silica hydrogel.
- The unreacted cores of the glass particles remain as a filler

***Clinical application.*** The set material needs to be protected from salivary contamination for several hours. Conventional GICs are relatively technique-sensitive with regard to mixing and insertion procedures are very sensitive to desiccation (at the beginning) and hydration (at the end) during setting.

#### **Conventional Glass ionomers**

**Glass ionomers** have the same favorable characteristics:

1. Ability to adhesion to tooth structure, this is mechanism of chemical bonding between the carboxyl group of the polyacid and the calcium in the tooth structure.
2. They release fluoride into the surrounding tooth structure
3. Yielding a potential anti- cariogenic effect
4. Possess a favorable coefficient of thermal expansion and may help reduce microleakage

##### **Indications**

1. Restoration of teeth with root-surface caries (anti-cariogenic quality and adhesion to dentin).
2. Other restorations in patients exhibiting high caries activity.
3. For restorations in patients with xerostomia
4. For permanent cementation of crowns.
5. In Sandwich technique

***Resin-Modified Glass Ionomers (RMGI).***(RMGI) were developed to improve the physical properties and esthetic qualities of conventional glass ionomer cements. RMGIs are easier to use, possess better strength, wear resistance, and esthetics than conventional glass ionomers. Fuji II LC, Vitremer (3M ESPE), Vitrebond (3M ESPE) indicated principally for non-stress-bearing areas. Best indicated: Class V restorations, small approximal

anterior lesions, in minimum intervention dentistry, ART technique, liner/base applications. Fuji IX is preferred for larger areas of dentin replacement.

### **Classification**

Most practical classification of the GICs is on their clinical usage

1. Type I GICs are the luting cements, characterized by low film thickness and rapid set;
2. Type II GICs are restorative cements:
  - sub-type 1 GICs are aesthetic cements (available in both conventional and resin-modified presentations)
  - sub-type -2 GICs are ‘reinforced’ , are not necessarily stronger than Type II-1 products, they are more wear-resistant.
3. Type III GICs are the lining cements and fissure sealants, characterized by low viscosity and rapid set.

Ketac-Molar (3M/Espe), Chemflex (Dentsply), Fuji IX and Fuji IX GP (GC) - high viscosity GICs are promoted principally for small cavities in deciduous teeth, temporary restorations, liner/base applications, and in the Atraumatic Restorative Treatment’ (ART) technique, sandwich technique.

### **Question 5. Basic Concepts of Adhesion.**

Adhesion is state in which two surfaces are held together by interfacial forces which may consist of valence forces or interlocking forces or both.

In dentistry bonding of resin-based materials to tooth structure is a result of four possible mechanisms: mechanical, adsorption, diffusion, a combination of the previous three mechanisms.

I. Adhesive restorative techniques currently are used to accomplish the following: Restore Class I, II, III, IV, V, and VI carious or traumatic defects

II. Change the shape and the color of anterior teeth (e.g., with full or partial resin veneers)

III. Improve retention for porcelain-fused-to-metal (ceramometal) or metallic crowns Bond all-ceramic restorations

IV. Seal pits and fissures Bond orthodontic brackets

V. Bond periodontal splints and conservative tooth- replacement prostheses

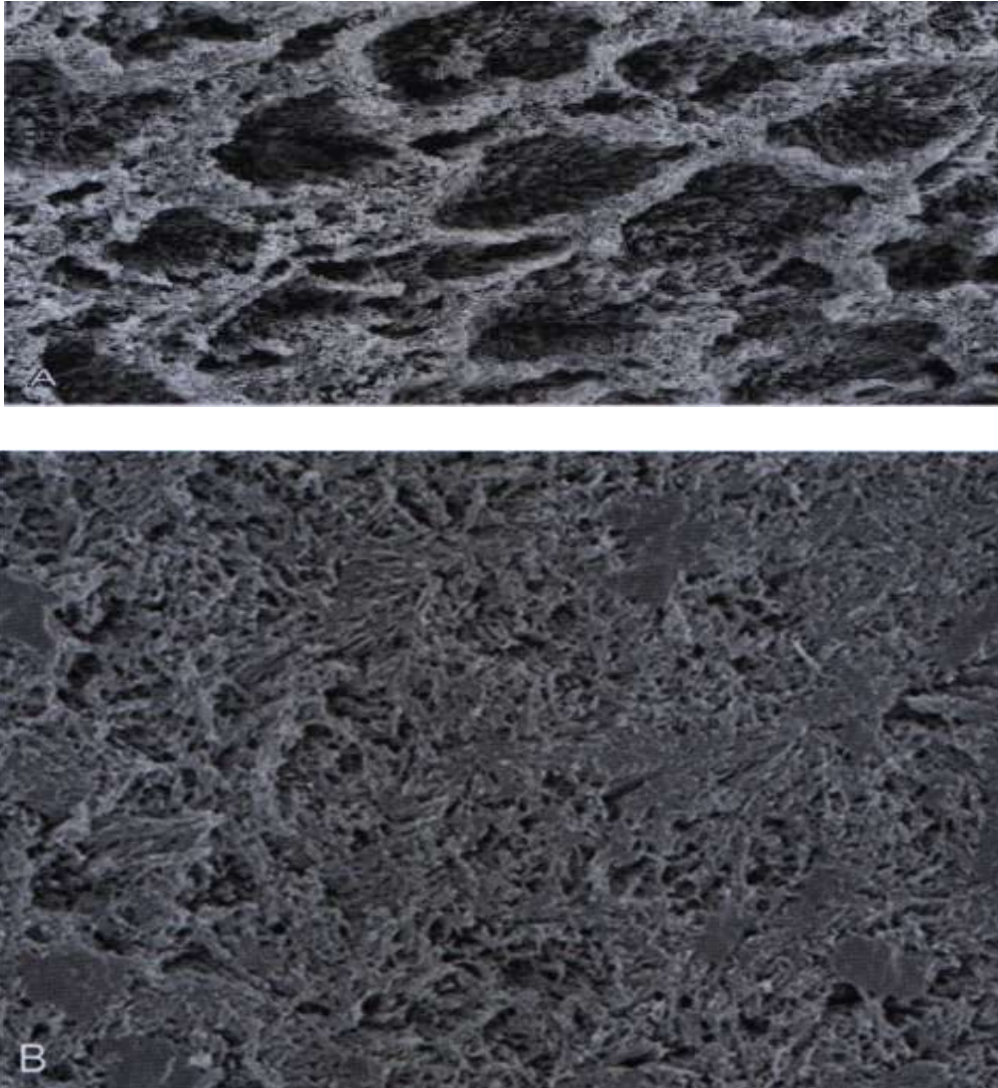
VI. Repair existing restorations (composite, amalgam, ceramic, or ceramometal)

**Enamel Adhesion.** Since Buonocore’ s introduction of the acid-etch technique, many dental researchers have attempted to achieve methods for reliable and durable adhesion between resins and tooth structure.

Acid-etching transforms the smooth enamel into an irregular surface and increases its surface free energy. When a fluid resin-based material is applied to the irregular etched surface, the resin penetrates into the surface,

aided by capillary action. Monomers in the material polymerize, and the material becomes interlocked with the enamel surface.

The acid-etch technique has revolutionized the practice of restorative dentistry. Bonding techniques allow more conservative tooth preparation, less reliance on macromechanical retention, and less removal of unsupported enamel.



*Figure 59. Scanning electron micrograph of enamel etched with 35% phosphoric acid for 15 seconds*

**Dentin Adhesion.** Adhesive materials can interact with dentin in different ways—mechanically, chemically, or both. The importance of micromechanical bonding, similar to what occurs in enamel bonding. For composites Dentin adhesion relies primarily on the penetration of adhesive monomers into the network of collagen fibers left exposed by acid etching. For Glass ionomer cements Dentin adhesion relies on the chemical bonding between polycarboxylic groups and hydroxyapatite.

**Question 6. Advantages and disadvantages of different types of restorative materials.**

**Table 40. Advantages and disadvantages of different types of restorative materials**

<b>Amalgam</b>	<b>Composites</b>	<b>Glass ionomers</b>	<b>Resin ionomers</b>
<b><i>Principal uses</i></b>			
Dental fillings	Aesthetic dental fillings	Small non-load fillings	Small non-load fillings
Heavily loaded posterior restorations	Veneers	Cavity liners	Cavity liners
		Cements for crowns and bridges	Cements for crowns and bridges
<b><i>Overall durability, fracture resistance, wear resistance</i></b>			
Good to excellent durability in large load bearing restorations	Good durability in small to moderate restorations	Moderate to good durability in non load-bearing restorations; poor in load-bearing	Moderate to good durability in non load-bearing restorations; poor in load-bearing
Brittle, subject to chipping on filling edges; good bulk strength in large high-load restorations	Moderate resistance to fracture in high load restorations	Low resistance to fracture	Low resistance to fracture
High resistance to wear	Moderate resistance to wear	High wear on chewing surfaces	High wear on chewing surfaces
<b><i>Cavity preparation and clinical consideration</i></b>			
Require removal of tooth structure	Adhesive bonding permits removal of less tooth structure	Adhesive bonding permits removal of less tooth structure	Adhesive bonding permits removal of less tooth structure
Tolerant to wide range of clinical conditions	Requires well-controlled field of operation	Requires well-controlled field of operation	Requires well-controlled field of operation
Moderately tolerant to moisture during placement	Very little tolerance to moisture during placement	Very little tolerance to moisture during placement	Very little tolerance to moisture during placement

<i>Leakage and recurrent decay</i>			
Moderate leakage	Low leakage if properly bonded	Low leakage generally	Low leakage if properly bonded
Recurrent decay same as other materials	Recurrent decay depends on maintenance of tooth-material bond	Recurrent decay comparable to other materials	Recurrent decay comparable to other materials
		Fluoride release may be beneficial	Fluoride release may be beneficial

Table 41. Annual failure rates of dental restorations

<i>Material</i>	<i>Age at replacement</i>	<i>Annual failure rate</i>
Resin-based composites	8 years	2,3%
Poly-acid modified composites	7 years	3,5%
Resin-modified glass ionomers	2 years	3,1%
Glass ionomers	4 years	7,6%
Amalgam	10 years	2,2%

### Tests to the topic

#### 1. Indicate requirement for work with composites:

- Dryness; isolation of operation field; absence of allergic reaction to resin.
- Wet cavity.

#### 2. What are advantages of microphylls?

- Viscosity.
- Aesthetics, good polishability, high elasticity coefficient.
- High strength.

#### 3. What are positive aspects of composites materials?

- Aesthetics.
- Low thermal conductivity.
- Flexible design, chemical resistance.
- Multi-purpose.
- All answers are correct.

#### 4. What are negative properties of composite materials?

- Polimerazation shrinkage, hydrophility.
- Formation of dispersion layer.
- Allergic reaction of resin.
- Viscosity.

- e. All answers are correct.

**5. Name the complications of polymerization shrinkage**

- a. Secondary caries; filling discoloration.
- b. Destroying of the marginal wall of a restoration.
- c. Microcracks, microleakage.
- d. Hypersensitivity.
- e. All answers are correct.

**6. What is direction of polymerization shrinkage in composites during polymerization?**

- a. Towards the center of restoration.
- b. Towards the light source.
- c. Towards the dental pulp.

**7. Disadvantages of classic glass-ionomer cements (gic) are**

- a. Sensitivity to moisture in last stages of curing.
- b. Sensitivity to overdrying.
- c. Mechanical instability.
- d. All answers are correct.

**8. Name the indications for glass-ionomer cements use:**

- a. Isolating liners for all types of filling materials; permanent filling for cavities of III, v classes of permanent teeth; permanent filling in cavities of I-V classes of temporary teeth; long delayed filling of cavities of I, II classes of permanent teeth.
- b. Permanent filling in cavities of I, II, IV classes of permanent teeth.

**9. What is the classical gic adhesion mechanism to hard tooth tissues?**

- a. Micromechanical.
- b. Macromechanical.
- c. Chemical.

**10. What is cavity configuration factor (c-factor)?**

- a. Ratio of the unbonded to the bonded surface area.
- b. Ratio of the bonded to the unbonded surface area.
- c. Coefficient of thermal expansion.



## **LESSON 17. MINIMAL INVASIVE METHODS OF OPERATIVE TREATMENT OF DENTAL CARIES**

The questions to be studied for the learning of the topic:

1. The minimal invasive methods of operative treatment of dental caries: objectives, features, advantages and disadvantages, techniques.
2. ART-technique, indications for use, definition.
3. Tunnel preparation, indications for use, methods of conducting.
4. Icon technology: indications for use, methods of conducting.

### **QUESTION 1. The minimal invasive methods of operative treatment of dental caries: objectives, features, advantages and disadvantages, techniques.**

**Minimally intervention dentistry (MID)** adopts a philosophy that integrates prevention, remineralisation and minimal intervention for the placement and replacement of restorations. The objective is tissue preservation (preferably by preventing disease and intercepting its progress), this means performing treatment with as little tissue loss as possible. It expresses a very precise excision of what has to be removed, without causing any damage to adjacent tissue. With the new techniques available (digital radiology with low radiation emission, diagnostic laser and the dental operative microscope) we can aim for both an early diagnosis and a minimally invasive therapy (ozone therapy, air abrasion, rotary instruments for micro preparation and the laser. The rules for dentistry were invented in the late 1800's by Dr. G.V.Black. The idea was to remove the possibility of further decay on the surface of the tooth already afflicted with caries. The act of making a restoration therefore usually involved the removal of a substantial amount of tooth structure, often several times more than was actually decayed. Hence, a lot of healthy tooth were destroyed in the process.

The minimally invasive approach in treating dental caries incorporates the dental science of detecting, diagnosing, intercepting and treating dental caries at microscopic level. This approach has evolved from an increased understanding of the caries process and the development of adhesive and biomimetic restorative materials. With minimally invasive dentistry, dental caries is treated as an infectious condition rather than an end product of it.

There are basic principles that must be applied to fulfill the description of minimal intervention dentistry:

1. Control the disease through reduction of cariogenic flora.
2. Remineralise early lesions. Perform minimal intervention surgical procedures, as required.
3. Repair, rather than replace defective restorations.

**Table 42. Characteristic of minimal invasive methods of operative treatment of dental caries**

<i>Features</i>	<ol style="list-style-type: none"> <li>1. Alternative preparation methods (manual, laser treatment, air abrasion).</li> <li>2. The use of special materials.</li> <li>3. The use of special tools.</li> <li>4. Possibilities of correction of clinically insolvent restorations.</li> </ol>
<i>Advantages</i>	<ol style="list-style-type: none"> <li>1. The minimum weakening of the structure of the tooth.</li> <li>2. Long-term and aesthetics.</li> <li>3. Preventive effect due to the release of fluorine material.</li> </ol>
<i>Disadvantages</i>	<ol style="list-style-type: none"> <li>1. High sensitivity to violation of the technique of execution.</li> <li>2. The high cost of services (laser processing, air abrasion).</li> </ol>

Minimal intervention operative dentistry is dependent on following factors:

1. Demineralization – Remineralization cycle
2. Adhesion in restorative dentistry
3. Biomimetic restorative material.

### **MI preparation techniques**

- Atraumatic restorative treatment (ART).
- Tunnel, box and slot preparation.
- Rotary - High/low speed bur.
- Sonic oscillation - SONICSYS micro.
- Chemomechanical – Carisolv.
- Kinetic - Air abrasion
- Hydro kinetic - Laser (CO, Er: YAG, Nd: YAG, etc.).
- Ozone technology – O<sub>3</sub>.
- Pit and fissure sealants and preventive resin restorations.
- Sandwich technique.
- Icon technology.

**Dental Materials Used for Minimally Invasive Treatment:** glass ionomer cement and adhesive dental materials.

### **Question 2. ART-technique, definition, indications for use.**

**Atraumatic Restorative Treatment(ART)** is the revolutionary technique of filling teeth without electricity, anesthetics or normal dental equipment. This revolutionary approach has been used extensively in developing countries. ART was developed by Dr. Jo Frencken DDS, MSc, PhD, and a dental researcher in the Netherlands. ART uses seven small hand

instruments to remove tooth decay and a filling material, within a fraction as hard as silver that chemically bonds to the tooth without pain. The powder when mixed as a filling is 28% fluoride. This fluoride is released up to eight years and decreases decay and gum disease substantially on the same side of the mouth as the ART filling. ART differs from traditional silver or composite fillings as a minimally invasive cavity preparation. Very little, if any, healthy tooth is removed in the ART technique. The amount of completely demineralized dentine (decay) removed dictates the size and extent of the ART cavity preparation. The traditional cavity size and extent are dictated by the removal of all completely demineralized and partially demineralized dentine, plus extension for prevention of cutting out all pits, fissures and grooves which further weaken the tooth.

The Atraumatic Restorative Treatment (ART) is a procedure based on removing carious tooth tissues using hand instruments alone and restoring the cavity with an adhesive restorative material. At present the restorative material is glass-ionomer. This procedure has been developed because millions of people in less-industrialized countries and certain special groups such as refugees and people living in deprived communities are unable to obtain restorative dental care. The ART approach enables treatment of cavities in teeth to be provided for people residing in areas where electricity is not available or, alternatively, in areas which have electricity, but where the community cannot afford expensive dental equipment.

Glass-ionomers are very useful dental restorative materials. In addition to its use as a restorative material, glass-ionomers can be applied in the very early stages of caries development. The glass-ionomer sticks to the tooth and halts or slows the progression of lesions, mainly because it slowly releases fluoride.

ART is, however, just one component of oral health care which must start with health promoting messages about a prudent diet and good oral hygiene using a fluoride containing toothpaste. Sealing pits and grooves in the chewing surfaces of teeth is another preventive action to consider. Removing carious tooth tissue with hand instruments alone, and restoring the cavity with an adhesive material - that is ART - will conserve as much tooth structure as possible and prevent further decay.

**Table 43. Advantages and Disadvantages of ART-technique**

<i>Advantages</i>	<ol style="list-style-type: none"> <li>1. Easily available inexpensive hand instruments are used rather than the expensive electrically driven dental equipment.</li> <li>2. As it is almost a painless procedure the need for local anesthesia is eliminated or minimized.</li> <li>3. ART involves the removal of only decalcified tooth</li> </ol>
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	<p>tissues, which results in relatively small cavities and conserves sound tooth tissues as much as possible.</p> <ol style="list-style-type: none"> <li>4. Sound tooth tissue need not be cut for retention of filling material.</li> <li>5. The leaching of fluoride from glass ionomer probably remineralizes sterile demineralized dentin and prevents development of secondary caries.</li> <li>6. The combined preventing and curative treatment can be done in one appointment.</li> <li>7. Repairing of defects in the restoration can be easily done</li> <li>8. It is less expensive and less time consuming.</li> <li>9. It enables to oral health workers to reach people who otherwise never would have received any oral health service.</li> </ol>
<b>Disadvantages</b>	<ol style="list-style-type: none"> <li>1. ART restorations are not long lasting. The average life is two years depending upon the rate of caries activity of the individual oral cavity.</li> <li>2. Because of the low wear resistance and low strength of the existing glass ionomer materials their use is limited.</li> <li>3. A relatively unstandardized mix of glass ionomer may be produced due to hand mixing.</li> <li>4. The continuous use of hand instruments over long period of time may result in hand fatigue.</li> <li>5. As fundamental principles of cavity preparation are not followed all oral health workers may not accept it.</li> </ol>

In order to carry out the Atraumatic Restorative Treatment approach in the field, the following essential instruments and materials are required.

**Table 44. Instruments and Materials for ART-technique**

<b>Instruments</b>	<b>Materials</b>	<b>Other</b>
Mouth mirror	Cotton wool roll	Examination gloves
Explorer	Cotton wool pellet	Mouth mask
Pair of tweezers	Clean water	Operating light
Dental hatchet	Glass-ionomer restorative material	Operation bed / headrest extension
Spoon excavator, small	liquid, powder and	Stool
Spoon excavator, medium	measuring spoon	Methylated alcohol
Spoon excavator, large	Dentine conditioner	Pressure cooker
Applier/carver	Petroleum jelly	Instrument forceps
Glass slab or paper	Wedge	Soap and towel
	Plastic strip	Sheet of textile
	Articulation paper	

mixing pad Spatula		
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### Question 3. Tunnel preparation, indications for use, methods of conducting.

Tunnel restoration is one of the methods of restorative dentistry, providing a conservative approach to the preparation of the cavity with the maximum preservation of healthy dental tissues. This method has been described by G. M. Jinks, 1963. It was proposed to use it for the restoration of the distal approximal surfaces of temporary second molars. Access to the carious lesion was carried out from the occlusal surface, tooth tissues were prepared below the marginal ridge, which was kept intact. The teeth were sealed with fluorine-containing silicate cement. This method was reproduced by Hunt and Knight as a more conservative approach than traditional cavity formation Black class II in the 1980s. The method was used to treat primary caries on the approximate surfaces of the posterior teeth. Materials for filling are glass ionomer cements and flowable composites.

This method can be applied to patients with low intensity and low risk of caries disease. Tunnel restoration is contraindicated in the case of a high risk of developing carious disease, the presence of tissue defects in the marginal ridge that have arisen before or after preparation of the carious cavity.

**Types of TP.** Some authors describe the *internal tunnel preparation*, which in reality is the treatment of cavities of class I according to Black. In such cases, the approximal enamel is preserved (macroscopically there is no cavity). *Partial tunnel preparation* extends to the approximal surface in a macroscopically observed cavity or to the area where the enamel is demineralized. Enamel is carefully smoothed around the defect. Its demineralized part is preserved, and later it will be adjacent to the filling. All demineralized enamel on the approximal surface is removed in the case of *complete tunnel preparation*.

Table 45. **Advantages and Disadvantages of tunnel preparation**

<b>Advantages</b>	<ol style="list-style-type: none"> <li>1. Preserves the marginal ridge - conservative approach</li> <li>2. Less potential for a restorative overhang</li> <li>3. Perimeter of the restoration is reduced, decreasing the potential for micro leakage.</li> <li>4. Potential for disturbance of the adjacent tooth is reduced</li> </ol>
<b>Disadvantages</b>	<ol style="list-style-type: none"> <li>1. Highly technique sensitive, demanding careful control of the preparation by the operator</li> <li>2. Angulations of preparation often passes close pulp</li> <li>3. Visibility is decreased and caries removal is more</li> </ol>

	uncertain
	4. Fragile marginal ridge - at least 2.5 mm apical to crest of the marginal ridge (Mount 1997)

### *Stages of TP*

1. Local anesthesia.
2. Applying cofferdam.
3. Tooth removal from plaque with polishing paste, brush and low-speed handpiece.
4. Access to the carious cavity on the aproximal surface of molars or premolars from the occlusal surface.
5. Further preparation of the carious cavity by spherical bor using a low-speed tip.
6. The applying of the matrix and its fixation with wedges.
7. Treatment of the cavity with air conditioning, included in the set of glass ionomer cements.
8. Mixing the glass ionomer cements.
9. The introduction of the glass ionomer cements in the cavity and condensation of the material.
10. Removal of the occlusal part of the material with spherical bor to a depth of about 1 mm (as soon as the glass ionomer cements hardens).
11. Etching enamel on the edges of the cavity on the occlusal surface phosphoric acid for 30 seconds.
12. Washing with water and drying the cavity on the occlusal surfaces, adhesive application, polymerization, introduction and polymerization of a composite material.
13. Removal of the matrix, if necessary - polishing of the glass ionomer cements on the approximal surface by strips.
14. Removal of the cofferdam, correction of occlusal contacts.
15. Instruction on oral hygiene, especially with regard to the regular use of floss.
16. Clinical assessment of the quality of the restoration (after 6 months) and radiological (in a year).

### **Question 4. Icon technology: indications for use, methods of conducting.**

**Icon** is an innovative product for the micro-invasive treatment of cariogenic lesions in proximal regions and on smooth surfaces and available in two variations:

***Icon Caries Infiltrant –Proximal*** is a specially developed hard tissue preserving treatment for incipient proximal caries.

***Icon Caries Infiltrant–Smooth Surface*** is a specially developed infiltration product for smooth surfaces and particularly well-suited to

remove white spots and treat incipient caries for orthodontic patients after bracket removal.

Table 46. **Characteristic of Icon technology**

<b>Indications</b>	<ul style="list-style-type: none"> <li>✓ Micro-invasive treatment of non-cavitated cariogenic enamel lesions (lesion depth up to D1).</li> <li>✓ Esthetic improvement of mild to moderate noncavitated fluorotic lesions (on smooth surfaces)</li> </ul>
<b>Contraindications</b>	<ul style="list-style-type: none"> <li>✓ For deeper seated lesions (D2 – D3) (see “Lesion depth classification”) with cavitated enamel (enamel defects).</li> <li>✓ In case of known allergies to any material component or existing contact allergies.</li> </ul>
<b>Side effects</b>	<ul style="list-style-type: none"> <li>✓ In some cases, contact allergies with similar composite products have been reported.</li> <li>✓ Contact of Icon-Etch with the oral mucosa causes a white surface coloration. This will subside after a few days.</li> </ul>
<b>Composition</b>	<ul style="list-style-type: none"> <li>✓ Icon-Etch: Hydrochloric acid, pyrogenic silicic acid, surface-active substances.</li> <li>✓ Icon-Dry: 99 % ethanol</li> <li>✓ Icon-Infiltrant: Methacrylate-based resin matrix, initiators, additives</li> </ul>
<b>Interaction</b>	Polymerization inhibiting substances such as products containing eugenol (e. G. Temporary cements) must not be used in combination with this material.



Figure 60. Lesion depth classification. Radiographic lesion depth classification according to bitewing images.

#### **PLEASE NOTE**

- Light-curing units should have an output of 450nm and should be checked regularly.
- The light intensity should be at least 800mW/cm<sup>2</sup>.
- Place the light unit as close to the material as possible.

- The etched surface must not be touched or contaminated with saliva or moisture until the treatment resumes. If contamination occurs after drying, re-etch for approx. 10 s and repeat drying with Icon-Dry.
- For best treatment results a sufficiently dry working area is critical. Therefore, appropriate measures for a complete isolation must be taken. Do not use rubber dams made from thermoplastic elastomers such as Flexi-Dam (Coltène Whaledent/Hygenic).
- Discard syringes after use.
- On superficially remineralized, mostly older white spots, the pseudo-intact surface layer may be fairly thick. In this case, a 2 minute etching step to remove the surface layer may not be sufficient and etching and rinsing may have to be repeated. The teeth are then dried with Icon-Dry and infiltrated.

**Proximal surface treatment:**

- ✓ The syringes included in the treatment unit contain sufficient material for treating two proximal lesions.
- ✓ Additional proximal surfaces can be treated consecutively during one visit.

**Smooth surface treatment:**

- ✓ The syringes included in the treatment unit contain sufficient material for treating two to three smooth surface lesions.
- ✓ Multiple smooth surface lesions can be treated at the same time.
- ✓ For smooth surface applications a liquid dam (Liquid Dam) can be used as well.
- ✓ Treating orthodontic patients after bracket removal: Unless white spots are treated early, i.e. 1 to 2 months after bracket removal, it is recommended to repeat the etching step. A third etching process is recommended if a white spot is still visible after Icon-Dry is applied.
- ✓ In total, a lesion can be etched up to three times for 2 minutes each with Icon-Etch.
- ✓ For topical cariogenic white spots an area of 2 mm beyond the lesion site is etched. At the discretion of the dentist the entire smooth surface area ought to be etched and infiltrated in case of large area white spots as they occur after bracket removal.

Table 47. **Recommended use**

<p><b><i>Proximal surface treatment with Icon Caries Infiltrant – Proximal</i></b></p>	<p><i>Preparation</i></p> <ol style="list-style-type: none"> <li>1. Before the start of treatment, clean the affected tooth and adjacent teeth. Remove any cleaning residue with water spray.</li> <li>2. A sufficiently dry working area is critical. Therefore, appropriate measures like rubber dam</li> </ol>
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	<p>or liquid dam must be taken. Please follow the manufacturer's specifications and instructions.</p> <p><i>Separation</i></p> <p>3. Introduce one of the enclosed dental wedges into the interdendum.</p> <ul style="list-style-type: none"> <li>– For better access to the proximal region the wedge handle can be bent or detached with a twisting motion.</li> <li>– In order to obtain sufficient separation of the teeth the wedges must be seated at an adequate depth in the interdendum.</li> <li>– A separation of approx. 50 µm is recommended to ensure a successful treatment. This can be done gradually: Insert the wedge to the point of resistance. Wait 3 to 5 s and gradually move the wedge deeper until sufficient separation is achieved.</li> <li>– Leave the wedge in the proximal space during the entire treatment procedure.</li> <li>– Alternatively, especially at very narrow proximal spaces, it is possible to separate the teeth with an orthodontic rubber ring.</li> </ul> <p><i>Uncovering the lesion body/</i> For best treatment results the hyper mineralized surface layer must be removed.</p> <p>4. Screw the Proximal-Tip onto the Icon-Etch syringe, and introduce the application tip into the interdendum. Be sure to align the green side of the ProximalTip with the area to be treated.</p> <ul style="list-style-type: none"> <li>– The material can only be dispensed on the green side of the application tip.</li> </ul> <p>5. Apply an ample amount of Icon-Etch onto the lesion site (1½ to 2 turns of the shaft). Let Icon-Etch set for 2 minutes. The etching gel will be activated by slightly moving the applicator. Remove excess material.</p> <p>6. Remove the application tip from the interdendum. Suction off Icon-Etch and rinse with water for a minimum of 30 s. Dry carefully with oil-free and water free air.</p> <p><i>Drying.</i> For best treatment results it is necessary to dry the lesion in-depth.</p>
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	<p>7. Screw the application tip onto the Icon-Dry syringe, apply an ample amount of material onto the lesion and allow to set for 30 s. Dry thoroughly with oilfree and water-free air.</p> <p><i>Infiltration.</i> The lesion can be penetrated in-depth by the Infiltrant after complete drying. Do not apply Icon-Infiltrant under direct operating light as this may cause the material to set prematurely.</p> <p>8. Screw a new Proximal-Tip onto the Icon-Infiltrant syringe, and introduce the application tip into the interdentium. Be sure to align the green side of the Proximal-Tip with the area to be treated.</p> <ul style="list-style-type: none"> <li>– The material can only be dispensed on the green side of the Proximal-Tip.</li> </ul> <p>9. Apply an ample amount of Icon-Infiltrant onto the lesion site (1½ to 2 turns of the shaft).</p> <p>10. Let Icon-Infiltrant set for 3 minutes. The Infiltrant will be activated by slightly moving the applicator. Add material if necessary.</p> <p>11. Remove the application tip from the interdentium. Remove excess material with dental floss.</p> <p>12. Light-cure Icon-Infiltrant from all sides for at least 40 s (total).</p> <p>13. Screw a new Proximal-Tip onto the Icon-Infiltrant syringe, repeat the application, and allow to set for 1 minute. Remove the application tip from the interdentium. Remove excess material with dental floss. Then light-cure from all sides for at least 40 s (total).</p> <p>14. If a second lesion needs to be treated repeat steps 1 through 13 for the affected site.</p> <p>15. Remove the wedge and rubber dam. Use polishing strips for the surface finish.</p>
<p><b><i>Smooth surface treatment with Icon Caries Infiltrant – Smooth Surface</i></b></p>	<p>Preparation</p> <p>1. Before the start of treatment, clean the affected tooth and adjacent teeth. Remove any cleaning residue with water spray.</p> <p>2. For best treatment results a sufficiently dry working area is critical. Therefore, appropriate</p>

	<p>measures like rubber dam or liquid dam must be taken. Please follow the manufacturer's specifications and instructions. Ensure that the treated lesion is completely accessible.</p> <p><i>Uncovering the lesion body</i>, For best treatment results the hyper mineralized surface layer must be removed.</p> <p>3. Screw the Smooth Surface-Tip onto the Icon-Etch syringe.</p> <p>4. Apply an ample amount of Icon-Etch onto the lesion site by turning the syringe shaft carefully, allow to set for 2 minutes and activate it by moving. Remove excess material with a cotton wad.</p> <p><i>Note</i>: Unless white spots are treated early, i. e. 1 to 2 months after bracket removal, it is recommended to repeat the etching step up to three times.</p> <p>5. Suction off Icon-Etch and rinse with water for at least 30 s. Dry with oil-free and water-free air.</p> <p><i>Visual Check</i>. For best treatment results it is necessary to dry the lesion in-depth.</p> <ul style="list-style-type: none"> <li>– In this step a preview of the final result is shown. When wetted with Icon-Dry, the whitish-opaque coloration on the etched enamel should diminish. If this is not the case repeat the etching step once or twice for 2 minutes each, and rinse and dry the teeth again (steps 3-5).</li> </ul> <p>6. Screw the application tip onto the Icon-Dry syringe, apply an ample amount of material onto the lesion, and allow to set for 30 s.</p> <p><i>Drying</i>. For best treatment results it is necessary to dry the lesion in-depth.</p> <p>7. After the visual check dry the lesion thoroughly with oil free and water-free air.</p> <p><i>Infiltration</i>. The lesion can be penetrated in-depth by the Infiltrant after complete drying.</p> <p>8. Screw a new Smooth Surface-Tip onto the Icon-Infiltrant syringe.</p> <ul style="list-style-type: none"> <li>– Do not apply Icon-Infiltrant under direct operating light as this may cause the material</li> </ul>
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	<p>to set prematurely.</p> <p>9. Apply an ample amount of Icon-Infiltrant onto the etched surface by turning the shaft.</p> <p>10. Allow Icon-Infiltrant to set for 3 minutes. The Infiltrant will be activated by slightly moving the applicator. Add material if necessary.</p> <ul style="list-style-type: none"> <li>– In case of deeper and larger defects the esthetic result can be improved by extending the exposure time up to 6 minutes.</li> </ul> <p>11. Remove excess material with a cotton wad and dental floss.</p> <p>12. Light-cure Icon-Infiltrant for 40 s.</p> <p>13. Screw a new Smooth Surface-Tip onto the Icon-Infiltrant syringe, repeat the application, and allow to set for 1 minute. Remove excess material with a cotton wad and dental floss, and light-cure for a minimum of 40 s.</p> <p>14. Remove the rubber dam. Use polishing cups (or similar) for the surface finish.</p>
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**One treatment unit includes:**

**Icon Caries Infiltrant – Proximal**

- 1 Icon-Etch syringe @ 0.3 ml fluid
- 1 Icon-Dry syringe @ 0.45 ml fluid
- 1 Icon-Infiltrant syringe @ 0.45 ml fluid
- Accessories

**Icon Caries Infiltrant – Smooth Surface**

- 1 Icon-Etch syringe @ 0.45 ml fluid
- 1 Icon-Dry syringe @ 0.45 ml fluid
- 1 Icon-Infiltrant syringe @ 0.45 ml fluid
- Accessories

**Tests to the topic**

**1. Indicate advantages of minimal intervention dentistry:**

- The minimum weakening of the structure of the tooth.
- Long-term and aesthetics.
- Preventive effect due to the release of fluorine material.
- All of the above.

**2. Indicate disadvantages of minimal intervention dentistry:**

- High sensitivity to violation of the technique of execution.
- The high cost of services (laser processing, air abrasion).
- Significant weakening of the tooth structure.

- d. Low esthetics of restoration.
- e. No preventive effect.

**3. Indicate the factors on which the minimal intervention dentistry depends on**

- a. Demineralization – Remineralization cycle.
- b. Adhesion in restorative dentistry.
- c. Biomimetic restorative material.
- d. All of the above.

**4. Indicate advantages of ART-technique:**

- a. Easily available inexpensive hand instruments are used rather than the expensive electrically driven dental equipment.
- b. As it is almost a painless procedure the need for local anesthesia is eliminated or minimized.
- c. ART involves the removal of only decalcified tooth tissues, which results in relatively small cavities and conserves sound tooth tissues as much as possible.
- d. Sound tooth tissue need not be cut for retention of filling material.
- e. The leaching of fluoride from glass ionomer probably remineralizes sterile demineralized dentin and prevents development of secondary caries.
- f. The combined preventing and curative treatment can be done in one appointment.
- g. Repairing of defects in the restoration can be easily done
- h. It is less expensive and less time consuming.
- i. It enables to oral health workers to reach people who otherwise never would have received any oral health service.
- j. All of the above.

**5. Indicate disadvantages of ART-technique:**

- a. ART restorations are not long lasting. The average life is two years depending upon the rate of caries activity of the individual oral cavity.
- b. Because of the low wear resistance and low strength of the existing glass ionomer materials their use is limited.
- c. A relatively unstandardized mix of glass ionomer may be produced due to hand mixing.
- d. The continuous use of hand instruments over long period of time may result in hand fatigue.
- e. As fundamental principles of cavity preparation are not followed all oral health workers may not accept it.
- f. All of the above.

**6. Tunnel preparation is applied in the case of:**

- a. Filling cavities with glass ionomer cements.
- b. Filling cavities with flowable composites.
- c. Filling of cavities of the Black class II, located between the equator and the occlusal surface.
- d. Filling of cavities of the Black class II, located between the equator and the neck of the tooth.
- e. Filling of cavities of the Black class III, located between the equator and the occlusal surface.
- f. Filling of cavities of the Black class III, located between the equator and the neck of the tooth.
- g. Teeth with microcracks in the edge ridge, or when there is less than 2 mm of healthy enamel under it.

**7. Indicate advantages of tunnel preparation:**

- a. Preserves the marginal ridge - conservative approach.
- b. Less potential for a restorative overhang.
- c. Perimeter of the restoration is reduced, decreasing the potential for micro leakage.
- d. Potential for disturbance of the adjacent tooth is reduced.
- e. All of the above

**8. Indicate disadvantages of tunnel preparation:**

- a. Highly technique sensitive, demanding careful control of the preparation by the operator.
- b. Angulations of preparation often passes close pulp.
- c. Visibility is decreased and caries removal is more uncertain.
- d. Fragile marginal ridge - at least 2.5 mm apical to crest of the marginal ridge (Mount 1997).
- e. All of the above.

**9. Icon technology is applied in the case of:**

- a. The initial type of caries is E1, E2, D1 in depth (where E1 is the external enamel lesion, D1 is one-third denture injury).
- b. Dentophobia, fear of local anesthesia.
- c. Chalky spots after orthodontic treatment (removal of braces).
- d. Initial caries in hard-to-reach areas (especially in the interdental spaces).
- e. All of the above.

**10. Indicate contraindications of Icon technology:**

- a. For deeper seated lesions (D2 – D3) with cavitated enamel (enamel defects).
- b. In case of known allergies to any material component or existing contact allergies.
- c. A developed form of the disease that has affected more than one third of the dentin.
- d. Treatment of children up to three years due to the presence in the composition of components that can damage too sensitive enamel.
- e. All of the above.

## LESSON 18. CARIES PREVENTION. FORECASTING METHODS OF DENTAL CARIES

The questions to be studied for the learning of the topic:

1. Types of caries prevention.
2. Methods of caries prevention.
3. Rational feeding.
4. Using of fluorides for caries prevention.
5. Oral hygiene.
6. Fissure sealants.

### Question 1. Types of caries prevention.

**Primary prevention** protects individuals against disease, such as immunisation, and prevention of the initiation of the disease, as in dietary advice and plaque control within dentistry. Primary prevention is aimed at keeping an individual and a population healthy and at minimising the risk of disease or injury. It is this stage that seeks to implement programmes, procedures or measures to prevent a disease, before it actually occurs. Programmes designed to prevent people from starting to use tobacco (primary prevention) or to help them quit if they have already started (secondary prevention) can help prevent oral cancer and periodontal diseases, and can also be an effective general health promotion strategy. Additionally, plaque control and diet are effective primary prevention methods for both the prevention of dental caries and periodontal disease. Other primary prevention methods include the provision of fissure sealants, water fluoridation and routine dental examinations and diagnostic radiographs.

**Secondary prevention** aims to limit the progression and effect of a disease at the earliest possible opportunity after onset. It refers to the cessation of the disease process and preventing its progressive activity to more advanced stages, as well as preventing the recurrence of the disease, with further primary prevention interventions and advice. Therefore, to stop disease progression and recurrence, once a condition has been recognised, actions are needed to control and eliminate the further spread of that condition. Removing carious tooth tissue and restoring structure and function at an early stage of the caries process can prevent tooth loss or the need for more extensive treatment. This intervention may be in the form of preventive resin restorations or the placement of more extensive restorations. Secondary prevention measures to diagnose and treat periodontal diseases include periodontal probing and diagnostic radiographs, professional removal of hard and soft deposits, and the local application of antimicrobial agents. Oral examinations of the soft tissues, in addition to obtaining a comprehensive



social history to assess past and present tobacco and alcohol use, are also effective measures for detecting oral cancer at its early, most treatable stages.

**Tertiary prevention** is concerned with limiting the extent of disability once a disease has caused some functional limitation. At this stage, the disease process will have extended to the point where the patient's health status has changed and will not return to the pre-diseased state. Tertiary disease prevention refers to the rehabilitation of an individual and, with respect to oral disease, the reestablishment and maintenance of the integrity of the oral cavity. In the dental caries process, tertiary prevention is aimed not only at restoring carious teeth but also must include further primary and secondary prevention in order to prevent further carious attack. This means that in addition to the placement of a restoration, the causes of caries must also be addressed as part of a clinically effective caries management programme. When considering periodontal disease, periodontitis can be treated by a variety of interventions and surgical procedures or by administering antimicrobial agents either locally or systemically, but again the etiology must be identified.

## **Question 2. Methods of caries prevention.**

All measures on the caries prevention it is possible to divide into state, social, medical, hygienical and educating. The system of state preventives includes measures on the health protection mother and child, guard of environment. Realization of the state system of measures of health protection population is directed foremost on the antenatal prevention of diseases, on forming and development of healthy child, on support of health of the grown man is basis of prophylactic direction of health protection in our country.

**The system of social measures** on the caries prevention is related to providing of healthy way of life is observance of the rational mode of labor, rest, scientifically grounded norms of feed, personal hygiene.

**To the hygiene measures** hygiene education of population from the questions of dentistry, control after the state of environment and feed, belong on the caries prevention.

**Educating measures** on the caries prevention are taken to diffusing learning about the healthy way of life, inoculating of skills of care of organs of cavity of mouth and support them in the healthy state. These tasks are decided by doctors, middle medical personnel.

**Medical measures** on the caries prevention are directed for development and introduction etiologic and the nosotropic grounded facilities and methods of influence on an organism and organs of cavity of mouth for the increase of their firmness to the caries, and also on the decline of cariogenicity of unfavorable factors of environment on the cavity of mouth.

Table 48. **Teeth caries prevention (Udovits'ka, 1987)**

<b><i>With medications</i></b>		<b><i>Without medications</i></b>	
<i>Exogenous</i>	<i>Endogenous</i>	<i>Exogenous</i>	<i>Endogenous</i>
Rational Hygiene	Strengthening somatic health	Medications of the fluorine, calcium, phosphorus.	Applications of or rinse with fluorine-containing solutions
The intensive mastication	Chronic diseases treatment	Vitamins	Using of remineralization medications
Minerals from water; tea	Rational diet	Immunomodulators and medications increasing general organism resistance	Pressurizing of fissures

The most important methods of caries prevention are rational feeding, using of fluoride in different forms and dosages, methods of removing plaque, fissure sealants and strengthening of somatic health.

### **Question 3. Rational feeding.**

A balanced, nutritious diet is essential in preventing tooth decay and gum disease. Everyday food ration should include:

- *Fruits and vegetables.* Combined, these should cover half of day ration.
- *Grains.* At least half of the grains should be whole grains, such as oatmeal, whole wheat bread and brown rice.
- *Dairy.* Choose low-fat or fat-free dairy foods most often.
- *Protein.* Make lean protein choices, such as lean beef, skinless poultry and fish. Protein choices also should include eggs, beans, peas and legumes.

### **Foods That May Harm Dental Health**

Empty calories foods such as candy (especially hard or sticky candies like lollipops, mints, taffy and caramel), sweets like cookies, cakes and muffins, and snack foods like chips are a cause for dental concern, not only because they offer no nutritional value, but because the amount and type of sugar that they contain that can adhere to teeth. The bacteria in the mouth feed off these sugars, releasing acids, and that's what leads to tooth decay. Sugar-containing drinks are soda, lemonade, juice and sweetened coffee or tea (iced or hot) are particularly harmful because sipping them causes a constant sugar bath over teeth, which promotes tooth decay.

Acidic foods like tomatoes and citrus fruits can have acidic effects on tooth enamel too, so patient should eat them as part of a meal, not by

themselves. Dried fruits, including raisins, are also good choices for a healthy diet, but since they are sticky and adhere to teeth, the plaque acids that they produce continue to harm teeth long after you stop eating them.

### **Foods That May Benefit Dental Health**

Cheese, milk, plain yogurt, calcium-fortified tofu, leafy greens and almonds are foods that may benefit tooth health thanks to their high amounts of calcium and other nutrients they provide. Protein-rich foods like meat, poultry, fish, milk and eggs are the best sources of phosphorus. Both of these minerals play a critical role in dental health, by protecting and rebuilding tooth enamel.

Fruits and vegetables are good choices for a healthy smile since they are high in water and fiber, which balance the sugars they contain and help to clean the teeth. These foods also help stimulate saliva production, which washes harmful acids and food particles away from teeth and helps neutralize acid, protecting teeth from decay. Plus, many contain vitamin C (important for healthy gums and quick healing of wounds) and vitamin A (another key nutrient in building tooth enamel).

Hands down, water is particularly fluoridated, water is the most tooth-friendly beverage.

### **Snacking**

For dental health, it's recommended that people limit eating and drinking between meals. Of course, sometimes eating between meals must happen. Unfortunately, most people choose foods like sweets and chips for snacks; foods that harm teeth by promoting tooth decay. Products recommended for snacking are cheese, yogurt, fruits, vegetables or nuts.

### **Question 4. Using of fluorides for caries prevention.**

Preparations of fluorine now are basic facilities of teeth decay prevention. The mechanism of protective action of fluorine on enamel consists in the assistance to the delay of phosphoric-calcium connections in an organism and processes of remineralization of hard fabrics of tooth and also braking of activity of bacterial enzymes in the cavity of mouth and dental deposit.

Modern information shows that the favourable action of fluorine is predefined by a few mechanisms:

1. Fluorine, uniting from a hydroxide apatites enamel, substituting for OH – groups, forms a fluorapatites, doing an enamel more strong and more proof to the action of acids. This connection reduces permeability of enamel.
2. The mechanism of anticarious action of fluorides is related also to their oppressive influence on growth and exchange of matters of microflora of cavity of mouth.

3. Connections of fluorine in saliva inhibit the transport of glucose in the cages of pathogenic bacteria and formations of for cellular polysaccharidess, which form the matrix of dental deposit.

4. Fluorides violates absorption of microorganisms on-the-spot cages of tooth, absorb albumins of saliva, glycoproteins, as a result of what prevent growth of dental name-plate.

5. And finally, at internal introduction the fluorides normalize an albuminous and mineral exchange.

Fluorides present in enamel and in the dental deposit catalysis «proceeding» in the early carious defeats due to remineralization of crystals of enamel, multiplies the size of crystals of hydroxide apatites.

From modern international data days even receipts of fluorides are distributed thus:

- ✓ very low = 0,1-0,6 milligrams;
- ✓ low =0,7-1,4 milligram;
- ✓ optimum = 1,5-4,0 milligrams;
- ✓ high (impertinent fluorosis) = 5-12 milligrams;
- ✓ ever-higher = 20 milligrams and more (at treatment of osteoporosis of bones by fluorines preparations).

The amount of fluorine in an organism depends on his maintenance in water and food products.

### **Endogenous fluoride prevention**

A specific endogenous prevention provides for:

1. Fluorination of drinking-water
2. Fluorination milk
3. Fluorination salts

***Fluorination of water.*** One of the acknowledged methods of caries prevention there is fluorination of drinking-water is controlled addition of connections to the fluorine to water of sources of water-supply with the purpose of leading to the concentration of ions of fluorine in a drinking-water to the level, which is sufficient for the effective teeth decay prevention and at the same time does not have an unfavorable influence on functional possibilities of organism of man, physical development and health of population.

Presently about 5% all population of earth (approximately 260 million persons) drink fluorination water. In spite of numerous objections of opponents of fluorination, the presence of undesirable effects is not well-proven, and although every objection must be explored, safety of fluorination of water can be considered set.

The optimum concentration of fluorine in a drinking-water is 1,0 mg/l. Fluorination of drinking-water allows get reduction of increase of permanent teeth decay on 40-50% - on 50-60%.

**Fluorination of milk.** The use of fluorinating milk is the alternative and effective method of prophylaxis. Milk a long ago brings over to itself attention of researchers on a number of reasons, so as:

- it is the necessary component of feed, especially in the first years of life;
- owns valuable nourishing properties necessary for organism;
- it is the basic source of calcium and phosphorus, fabrics of bones and teeth necessary for the structure;
- contains the high level of calcium, phosphorus and lactose which laminates carbonhydratess also.

Similar composition allows to milk to bring in the payment in the process of remineralization of enamel of teeth and in its defence.

For successful introduction of method of fluorination of milk certain terms are needed:

- high dental morbidity of population is in a region;
- low maintenance of fluorides is in a drinking-water;
- absence of other sources of system receipt of fluorides.

For fluorination of milk more frequent fluoride of sodium is used. Technology of fluorination of milk is simple and does not present difficulties.

The amount of fluoride, which must be added to milk, is guilty to take into account age of child and receipt of fluoride from other products and water.

At the use of fluorinating milk it is necessary to adhere to the followings recommendations:

- effectively to use this method at children from 3 to 12 years
- daily a child must use a 1 glass of milk from 0,5 mg of fluoride
- during a year a child must drink milk not less 250 days.

**Fluorination of salt.** Fluoride in salt is available in several countries (in Belarus too). Recommended fluorides concentration in salt is 250 – 350 mg/kg. For caries prevention it is necessary to use salt with fluorine every day.

### **Exogenous fluoride prevention**

At the use of fluorine it is needed to take into account the following:

1. The concentration of fluorides not must exceed for a local prophylaxis 1-2% (calculating on a fluorine), as with the increase of concentration efficiency does not grow.
2. Efficiency of influencing is conditioned by their concentration in the free ionized kind.
3. It is necessary to take into account in this connection fastening possibility fluoride ions with the ions of calcium.
4. Fluorides is appointed taking into account maintenance of fluorine in a drinking-water and climatic factors.

***Tooth-pastes.*** The most commonly used product containing fluoride is fluoride toothpaste, which dominates on the toothpaste market. The amount of fluoride in toothpaste varies between 250 ppm and 2 500 ppm. Usually recommended concentration for adults is 1 500 ppm. Strong scientific evidence shows that the use of fluoride toothpaste has an effect on the prevention of caries in the permanent teeth of children and young adults. This effect is dose dependent, ie, toothpaste with higher fluoride concentration, 500 ppm fluoride, yields better effect than toothpaste with 1 000 ppm. The effect of fluoride toothpaste on primary teeth has been insufficiently assessed, as have the effects in adults and elderly people. However, nothing suggests that preventive effects would not be found in these age groups.

***Fluorine containing solutions for the independent use.*** The wide use in the prophylaxis of caries was found by solutions with the low concentrations of fluoride.

Amount of rinses makes:

- by a 0,05% solution -1 once on a day
- by a 0,1% solution -1 once in a week
- by a 0,2% solution – 1 one time in two weeks

For the improvement of cooperation of fluorine with an enamel preliminary it follows well to clean teeth and rinse a mouth by alkaline water for the change of pH environment. The rinse lasts 1-3 minutes. After it a mouth is rinsed by clean water.

Application of rinses by solution of fluorine sodium gives reduction to the caries 30%.

***Fluorine containing varnishes.*** One of widespread facilities of local prophylaxis there are varnishes which use for the prolonged period of influencing of fluoride on enamel. They form tape adjoining to the enamel, and which remains on teeth during a few hours and in fissures a few days and even weeks.

A fluorine – varnish shows itself composition of natural resins of vegetable origin. At the market presented: „Ftorlac” (Kharkiv), varnish „Duraphat”, „Belac” (Vladmiva).

A method is given recommended at the moderate or high level of intensity of caries of teeth, to the people with the high risk of origin of caries. Frequency of causing of varnish is 2-4 times per a year, depending on activity of caries.

Method: the surface of teeth is purged from the deposit and is dried out. Then by the special brush varnish is inflicted by a skim on the surface of tooth. At the same time it is possible to cover all teeth on one jaw or 3-5 teeth. For getting dry of varnish it is needed about 2-3 minutes. It is possible to dry the varnish by the compressed air. After coverage of teeth by fluorine varnish it is impossible to use the meal of 1-3 hours and in future the desired

only spoon-meat. It is not recommended to clean teeth 24 hours. Varnish is contained on-the-spot tooth not less 12 hours and for this time his ions penetrate on a depth to 100 mcm of healthy enamel.

To cover teeth by varnish it follows depending on activity of cariosity: at a 1 degree - 2 times per a year, at 2 — 4 times per a year, at 3 — from 6 to 12 times per a year. Triple coverage of teeth is recommended with an interval 1-2 days. It is set that in a year after application of fluorine containing varnish second caries of teeth goes down on the average on 50%.

***Fluorine containing gels and solutions for the professional use.*** Gels and solutions of fluoride of sodium 1% and 2% are used for appliques and electrophoresis. A doctor-dentist conducts procedure in the conditions of policlinic. Remineralization action of gels is based on diffusion of matters from gel in saliva and from her in the enamel of tooth.

Method: teeth preliminary clear, dry out and impose the wadding tampon well moistened by solution of fluoride of sodium on 3- 5 minutes. At first assess the masticatory surfaces of teeth, and then – labial and cheeks on both jaws. If gel is used, he is inflicted by heated by a brush and give to dry out. After procedure does not recommend eat and drink during 2 hours.

As a rule conduct 3-5 appliques by solution twice on a year and 2-6 appliques by gel on a year.

*Using of fluorides by a spoon*

1. To choose the spoon of the proper size. It must be the covered is all dental row, including the areas of retraction and it follows to provide access of gel to the contact with the structure of teeth. The ends of spoon (peripheral areas) must be closed in order that gel did not flow down in the cavity of mouth sick. Ideally spoons befit with coverage from the made foam material, as they fit snugly dental row of patient and allow to gel to achieve all surfaces.
2. To place gel in a stretcher.
3. To insert a spoon in the mouth of patient.
4. To insert between the spoons of saliva ejector, making sure, that to the patient comfortably (at this method for balance of bite from opposite sides necessary wadding rollers)
5. To bring a spoon out of mouth sick.
6. To ask a patient to spit out immediately after the delete of spoon. After procedure, at a necessity the delete of superfluous fluoride, to apply the intensive sucking.
7. To warn a patient that during 30 minutes after procedure it is impossible to eat or drink.

***Application of fluorine containing disks.*** Fluorine containing disks (paper and paraffins) are produced in packing for 10 things. The expense of

material is a 1 disk on procedure. The disk of «Ftorglicofoskal» contains the followings ingredients:

- neurosin - 8-16 g,
- fluoride of sodium - 0,5-2 g,
- superficial matters - 0,5-2,0 g,
- beeswax - 4,5-6,5 g,
- paraffin.

A disk is fixed in an angular tip by a mandrel. A fluorine is rubbed in hard fabrics of tooth on minimum speed with the use of three types of motions: recurrently-forward, up-down, circles. As usual, before treatment by fluorine containing disks the professional hygiene of cavity of mouth is conducted, whereupon by disks at first the vestibular surfaces of all teeth of maxilla are processed from left to right, then lower from right to left. After it the palatal surfaces of teeth of maxilla and languages surfaces of teeth of lower jaw are processed, farther are masticatory surfaces of teeth of overhead and lower jaws with the use of only circular motions clockwise. It is recommended 2-3 multiple treatment of teeth with an interval 1-2 days, in a year 2-4 courses. In practice of therapeutic dentistry fluorine containing disks found large popularity not only at the prophylaxis of teeth decay but also at treatment of hyperesthesia of hard fabrics of teeth.

### **Question 5. Oral hygiene.**

Good oral hygiene involves the removal of biofilm from the surface of the teeth, resulting in a mouth that has an absence of or few occurrences of bad breath, teeth that looks relatively clean and debris-free and healthy gums that are pink and do not bleed easily when brushing or flossing. The aim of practicing good oral hygiene is to modify the oral microflora so as to maintain an oral environment of healthy gums and teeth, prevent dental caries, periodontal disease and halitosis.

#### **Measures to practise good oral hygiene**

***Toothbrushing.*** There are many toothbrushing methods such as the Bass, Stillman's, Fones, Charter's, horizontal, vertical and scrub and roll method.

The main recommendations on toothbrushing:

- ✓ Brush the upper and lower set of teeth separately.
- ✓ When brushing the outer surfaces of the teeth, place the toothbrush at an angle of 45 degrees against the gumline and using a sweeping action, gently move the brush against the gumline, using a vibratory motion.
- ✓ When brushing the occlusal surfaces, use short forward and backward strokes.
- ✓ Similarly, when brushing the palatal or lingual surfaces of the teeth, tilt the toothbrush at an angle of 45 degrees against the gumline.



- ✓ An average time taken for brushing teeth is 2-3 minutes.

**Frequency of brushing.** Due to a variation in individual's preferences and habits, it is difficult to set the number of times one should brush his teeth. Instead, the emphasis should be placed on the effectiveness of biofilm removal from the surfaces of the teeth rather than on the number of brushings. It is recommended that a minimum of two brushings a day plus interdental cleaning is performed to remove the biofilm, hence preventing dental caries and bad breath. The longer the bacteria in the mouth is left undisturbed, the greater the pathologic potential of the bacteria. It is recommended to brush one's teeth before going to sleep because bacteria thrive in the warm, dark and moist conditions of the oral cavity. Based on research findings, the amount and rate of saliva flow changes in the 24-hour period. The normal flow rate of saliva in the day is about 0.3ml/min while at night, during sleep, the flow rate is about 0.1ml/min. Thus at night, the benefits of saliva are minimised in the controlling the bacterial population and its detrimental effects on the dentition.

**Type of Toothbrush.** A suitable toothbrush should be chosen based on many factors. Firstly, the size of the handle of the toothbrush should be chosen based on the age and the dexterity of the individual while the size of the head of the toothbrush should be based on the size of the individual's mouth. Toothbrushes with small heads are recommended because it enables easier access to the tooth surfaces at the back of the mouth. The bristles of the toothbrush should be end-rounded nylon or polyester filaments that are smaller than 0.009 inches in diameter and should be classified under 'soft' by the acceptable international industry standards (ISO). The toothbrush should be regularly replaced every 3 months or when it begins to show wear.

**Replacement of old toothbrushes.** It is recommended that toothbrushes should be replaced every 3 months. This is because toothbrush bristles that are worn down are less effective in removing biofilm and plaque from the surface of the teeth. The frequency of toothbrush wear varies between individuals, ranging from as early as two weeks to as long as 6 months. Hence, it is recommended to replace toothbrushes every 3 months or when it begins to show wear. Indicators of toothbrush wear include spreading, bending and curling of the bristles.

**Electric toothbrush.** An electric toothbrush is a modified toothbrush that runs on electric power to make rotation oscillations in the head. It is ideal for individuals suffering from arthritis and carpal tunnel syndrome. Based on studies done, electronic toothbrushes with rotational oscillations remove more plaque than manual toothbrushes and reduced the occurrence of gingivitis in the long term.

**Toothpaste.** Usage of fluoride toothpaste can help to prevent caries to a large extent. It was estimated that brushing with a fluoride toothpaste twice a

day reduced carious lesions by 90%. A combination of both cleaning with a toothbrush and using fluoride toothpaste is most optimal. It is also suggested that may be a cumulative effect of brushing the teeth with fluoride toothpaste. The concentration of fluoride present in toothpaste should be chosen based on age as well as the risk of caries. A fluoride concentration of 1100-1500 ppm in toothpaste is recommended for individuals above the age of seven. Rinsing the toothpaste with large amounts of water is also not advised because it removes a significantly larger amount of fluoride from the mouth. It is instead, recommended to clean off excess toothpaste using a wet toothbrush and spitting out as much toothpaste as possible. Proper toothbrushing alone, without the use of fluoride toothpaste, is still effective in removing plaque and biofilm from the surfaces of the teeth and oral hygiene is not compromised. However, it is recommended that fluoride toothpaste is used because fluoride helps to prevent carious lesion formations and remineralizes initial carious lesions.

***Interdental Cleaning.*** The removal of plaque on the interproximal surfaces is important in the prevention of caries. However, because the toothbrush is relatively ineffective in removing interproximal plaque, interdental cleaning methods, such as flossing, should be performed to maintain good oral hygiene.

***Flossing*** is one of the most commonly used methods of interproximal cleaning and according to the American Dental Association, an estimate of 80% of the interdental plaque can be removed by flossing.

1. Using a floss that is roughly 16 inches long, twirl most of the floss around the middle fingers, leaving a short length of about 1 to 2 inches to floss.
2. Hold the floss tightly between index fingers and thumb, slide it gently between the teeth, using a up-down movement towards the gum.
3. Curve the floss around the base of each tooth, making sure that it goes under the gumline.
4. Guide the floss back to the gum and repeat this twice.
5. Use a new section of the floss for each tooth, while winding the used sections to the middle finger of other hand.
6. Floss systematically, for example, from left to right, top to bottom, so as to not miss out on any tooth.
7. Some bleeding may be observed initially, however, after flossing regularly for a few days, the bleeding should stop.

There are various types of floss available. The nylon or multi-filament floss is available in the waxed and unwaxed form and in a variety of flavours. Because it is composed of many threads of nylon, it may shred at times, especially through tight contact points. The polytetrafluoroethylene (PTFE) or monofilament floss is only made up of one filament and is able to slide easily between teeth and is resistant to shredding. Superfloss has a stiff end

that acts as a floss threader to thread through tight areas. It also has a fuzzy tuft segment that collects plaque while flossing. The superfloss is very useful for flossing under bridges and for teeth with braces. There are also limitations in flossing in cases where there are embrasure spaces or when there is a loss of attachment. Due to the limitations in different individual's habits and ability to floss properly, some may find flossing difficult (especially through tight contact points).

***Interdental toothbrush.*** An interdental toothbrush is a small modified toothbrush structure that can be used to clean the spaces between teeth. It is useful in cases where there are gaps between the gingiva and the teeth or when there is orthodontic treatment. Interdental brushes can be used as a substitute for flossing and they are available in varying sizes to suit the size of the gaps between the teeth. To use an interdental brush, gently push it back and forth between the teeth but do not apply too much force.

***Tongue Cleaning.*** Tongue cleaning should be done regularly to remove food debris, fungi and bacteria build-up and dead cells from the surface of the tongue. Decaying bacteria produce volatile sulphur compounds that play a major role in halitosis/ bad breath. Tongue cleaning is not effective in controlling gingivitis or caries. However, by removing food debris and micro-organisms, tongue cleaning can help to contribute to overall oral cleanliness and reduce mouth odours. This is especially useful for patients suffering from xerostomia (dry mouth due to a lack of saliva), deep fissures and smokers. The top surface of the tongue can be cleaned using a tongue scraper/brush. Tongue scrapers are available in metal, plastic and many other materials. Tongue cleaning should be done using a proper tongue scraper and not a toothbrush.Procedure:

1. Place the arch of the tongue cleaner towards the posterior end of the dorsal surface of the tongue.
2. Pressing gently but firmly against the tongue, pull forward.
3. Repeat this process several times and complete the surface of the tongue.
4. Wash the tongue cleaner with water.
5. Some gag reflex may be experienced at the start.

### **Question 6. Fissure sealants.**

Fissure sealants are used on occlusal surfaces of the teeth. Occlusal surfaces have fissures that make tooth cleaning difficult and allow caries to start. The method involves applying a thin, very fluid, plastic material directly to the fissures. To retain the material to the tooth, its surface is pretreated with an acid that creates small pores in the enamel. The material fills the pores and there by mechanically bonds to the tooth surface, creating a smooth and even surface. It is essential to keep the tooth absolutely dry during treatment. If not, the pores can fill with saliva and the sealant attaches

poorly. Sealants must be applied soon after the tooth emerges to prevent early caries. Intact sealants have the potential to prevent caries on the occlusal surface. The method is not invasive and causes no pain. However, sealants are technically sensitive, and require continual checking and repair, or replacement if needed. Many studies have been reviewed to determine the preventive effects of fissure sealants composed of resin-based material. Many of the studies are outdated and deficient in study design and follow-up. Hence, there is only limited evidence in the literature that fissure sealants prevent caries in the short and long term. The literature offers insufficient evidence to assess other types of material used as fissure sealants. The scientific documentation also offers insufficient evidence for determining if fissure sealants have a preventive effect against caries in populations with low and high rates of caries.

***Strengthening of somatic health.*** It is assumed that under act of commons diseases the terms of forming and ripening of hard fabrics of tooth change in the first turn of enamel which does them less proof in relation to influencing of cariogenic factors. Practically the defeats of any organs and systems of organism are extrapolated on hard fabrics of teeth. That is why it is needed with the purpose of prophylaxis of dental diseases, on possibility, to treat be - what somatic pathology.

#### **Tests to the topic**

##### **1. What types of caries prevention do you know?**

- a. Primary prevention.
- b. Secondary prevention.
- c. Tertiary prevention.
- d. All answers are right.

##### **2. What are the most important methods of caries prevention?**

- a. Rational feeding.
- b. Using of fluoride in different forms and dosages.
- c. Methods of removing plaque.
- d. All answers are right.

##### **3. Everyday food ration should include:**

- a. Fruits and vegetables, grains, proteins.
- b. Cola, sweets, cakes.
- c. All answers are right.

##### **4. Endogenous fluoride prevention includes:**

- a. Fluorination of drinking-water.
- b. Fluorination milk.

- c. Fluorine containing solutions for the independent use.
- d. Fluorine containing varnishes.

**5.Exogenous fluoride prevention includes:**

- a. Fluorination of drinking-water.
- b. Tooth-pastes with fluorine.
- c. Fluorine containing varnishes.
- d. Fluorine containing solutions for the independent use.
- e. All answers are right.

**6. How many times it is recommended to cover teeth with fluorine containing varnishes?**

- a. Triple coverage with an interval 1-2 days.
- b. Double coverage with an interval 1-2 days.
- c. One coverage with an interval 1-2 days.
- d. All answers are right.

**7. How many times it is recommended to cover teeth with fluorine containing gels?**

- a. 2-6 appliques by gel on a year.
- b. 1-2 appliques by gel on a year.
- c. All answers are right.

**8. Usually recommended fluorine concentration in toothpastes for adults is:**

- a. 1500 ppm.
- b. 1000 ppm.
- c. 200 ppm.
- d. 2500 ppm.

**9. What amount of rinses with fluorine containing solutions for the independent use is recommended?**

- a. By a 0,05% solution -1 once on a day.
- b. By a 0,1% solution -1 once in a week.
- c. By a 0,2% solution – 1 one time in two weeks.
- d. All answers are right

**10. What are the main objects and tools using for oral hygiene?**

- a. Toothpaste.
- b. Toothbrush.
- c. Floss.
- d. Solutions and irrigator.

**LESSON 19. DISEASES OF HARD TISSUES OF TEETH, ARISING BEFORE ERUPTION (DEVELOPMENTAL DISORDERS):  
MOTTLED TEETH, HYPOPLASIA.**

**DISEASES OF HARD TISSUES OF TEETH OCCURRING AFTER THE ERUPTION (DENTAL ABRASION, GRINDING, EROSION OF THE TEETH)**

The questions to be studied for the learning of the topic:

1. Classification of noncarious lesions.
2. Anodontia.
3. Supernumerary teeth.
4. Abnormalities of size and form of teeth.
5. Mottled teeth.
6. Disturbances in tooth formation.
7. Enamel hypoplasia.
8. Tetracycline Discoloration.
9. Hereditary disturbances in tooth structure, not elsewhere classified.
10. Disturbances in tooth eruption.
11. Classification of noncarious lesions that appear after teeth eruption.
12. Excessive attrition of teeth.
13. Teeth abrasion.
14. Wedge defect.
15. Teeth erosion.
16. Teeth abfraction.
17. Pathological resorption of teeth.
18. Hypercementosis.
19. Ankylosis of teeth.
20. Trauma of teeth.

**Question 1. Classification of noncarious lesions.**

Noncarious tooth tissue loss is defined as surface loss due to a disease process other than dental caries (Pual A Brunton, Decision making in Operative Dentistry ). Although decay is the usual cause of tooth destruction necessitating operative procedures, it has been estimated that 25% of tooth destruction does not originate from a carious process.

According to the International classification of diseases there are 3 types of noncarious teeth lesions:

- disorders of tooth development and eruption (**K00**),
- embedded and impacted teeth (**K01**),
- other diseases of hard tissues of teeth (**K03**).

**K00 Disorders of tooth development and eruption**

K00.0 Anodontia, Hypodontia, Oligodontia

K00.1 Supernumerary teeth Distomolar, Fourth molar, Mesiodens, Paramolar, Supplementary teeth

K00.2 Abnormalities of size and form of teeth

Teeth Concrescence, Fusion, Gemination

Dens: evaginatus, in dente, invaginatus, Enamel pearls, Macrodonia, Microdonia, Peg-shaped [conical] teeth, Taurodonism, Tuberculum paramolare.

K00.3 Mottled teeth

Dental fluorosis

Mottling of enamel

Nonfluoride enamel opacities

K00.4 Disturbances in tooth formation

Aplasia and hypoplasia of cementum

Dilaceration of tooth

Enamel hypoplasia (neonatal, postnatal, prenatal)

Regional odontodysplasia

Turner tooth

K00.5 Hereditary disturbances in tooth structure, not elsewhere classified

Imperfecta Amelogenesis, Dentinogenesis, Odontogenesis

Dentinal dysplasia

Shell teeth

K00.6 Disturbances in tooth eruption

Dentia praecox

Natal, Neonatal tooth

Premature:

-eruption of tooth

-shedding of primary [deciduous] tooth

Retained [persistent] primary tooth

K00.7 Teething syndrome

K00.8 Other disorders of tooth development

Colour changes during tooth formation

Intrinsic staining of teeth

K00.9 Disorder of tooth development, unspecified

Disorder of odontogenesis

### **K01 Embedded and impacted teeth**

K01.0 Embedded teeth

K01.1 Impacted teeth

### **K03 Other diseases of hard tissues of teeth**

K03.0 Excessive attrition of teeth:

-approximal

-occlusal

K03.1 Abrasion of teeth:

- dentifrice
- habitual
- occupational
- ritual
- traditional

Wedge defect

#### K03.2 Erosion of teeth

Erosion of teeth due to:

- diet
- drugs and medicaments
- persistent vomiting
- idiopathic
- occupational

#### K03.3 Pathological resorption of teeth

Internal granuloma of pulp

Resorption of teeth (external)

#### K03.4 Hypercementosis

Cementation hyperplasia

#### K03.5 Ankylosis of teeth

#### K03.7 Posteruptive colour changes of dental hard tissues

#### K03.8 Other specified diseases of hard tissues of teeth

Irradiated enamel

Sensitive dentine

#### K03.9 Disease of hard tissues of teeth, unspecified

Depending on the occurrence of non-carious lesion of dental hard tissues are divided into two groups (M.I. Hroshykov, 1985):

- 1st - those that occur during follicular development tooth - hypoplasia, hyperplasia, fluorosis and hereditary lesions (dysplasia Kapdepona, imperfect Amelie and dentynohenez, marble disease, etc.)
- 2nd - damage arising after the eruption of the tooth - pathological tooth wear, wedge-shaped defects, necrosis, erosion of hard tissue, tooth hypersensitivity, trauma.

### **Question 2. Anodontia.**

**K00.0 Anodontia.** In dentistry, anodontia, also called anodontia vera, is a rare genetic disorder characterized by the congenital absence of all primary or permanent teeth. It is associated with the group of skin and nerve syndromes called the ectodermal dysplasias. Anodontia is usually part of a syndrome and seldom occurs as an isolated entity.

Anodontia is the congenital absence of teeth and can occur in some or all teeth (partial anodontia or hypodontia), involve two dentitions or only teeth of the permanent dentition (Dorland's 1998). Approximately 1% of the



population suffers from oligodontia. Many denominations are attributed to this anomaly: partial anodontia, hypodontia, oligodontia, the congenital absence, anodontia, bilateral aplasia.

Congenital absence of permanent teeth can present as hypodontia, usually missing 1 or 2 permanent teeth, or oligodontia that is the congenital absence of more than 6 teeth. The most common missing teeth are: lower third molars, upper lateral incisors, lower second premolars.

The congenital absence of at least one permanent tooth is the most common dental anomaly and may contribute to masticator dysfunction, speech impairment, aesthetic problems, and malocclusion (Shapiro and Farrington 1983).

*Treatment.* For the treatment of adontia are used orthodontic procedures and/or prosthetic replacement of missing teeth using dental implant technology or dentures.

### **Question 3. Supernumerary teeth.**

***K00.1 Supernumerary teeth.*** Supernumerary teeth (hyperdontia) is the condition of having teeth that appear in addition to the regular number of teeth. They can appear in any area of the dental arch and can affect any dental organ.

Supernumerary teeth can be classified by shape and by position. The shapes include the following:

- Supplemental (where the tooth has a normal shape for the teeth in that series);
- Tuberculate (also called barrel shaped);
- Conical (also called peg shaped);
- Compound odontoma (multiple small tooth-like forms);
- Complex odontoma (a disorganized mass of dental tissue).

When classified by position, a supernumerary tooth may be referred to as a mesiodens, a paramolar, or a distomolar.

The most common supernumerary tooth is a mesiodens, which is a malformed, peg-like tooth that occurs between the maxillary central incisors.



*Figure 61. Two mesiodens: supernumerary teeth in the midline between the upper central incisors.*

Fourth and fifth molars that form behind the third molars are another kind of supernumerary teeth.

**Causes.** Here is evidence of hereditary factors along with some evidence of environmental factors leading to this condition. While a single excess tooth is relatively common, multiple hyperdontia is rare in people with no other associated diseases or syndromes. Many supernumerary teeth never erupt, but they may delay eruption of nearby teeth or cause other dental or orthodontic problems. Molar-type extra teeth are the rarest form. Dental X-rays are often used to diagnose hyperdontia.

It is suggested that supernumerary teeth develop from a second tooth bud arising from the dental lamina near the regular tooth bud or possibly from splitting the regular tooth bud itself. Supernumerary teeth in deciduous (baby) teeth are less common than in permanent teeth.

#### **Question 4. Abnormalities of size and form of teeth.**

##### **K00.2 Abnormalities of size and form of teeth**

**Tooth fusion.** The phenomenon of tooth fusion arises through union of two normally separated tooth germs, and depending upon the stage of development of the teeth at the time of union, it may be either complete or incomplete. On some occasions, two independent pulp chambers and root canals can be seen. However, fusion can also be the union of a normal tooth bud to a supernumerary tooth germ. In these cases, the number of teeth is fewer if the anomalous tooth is counted as one tooth.



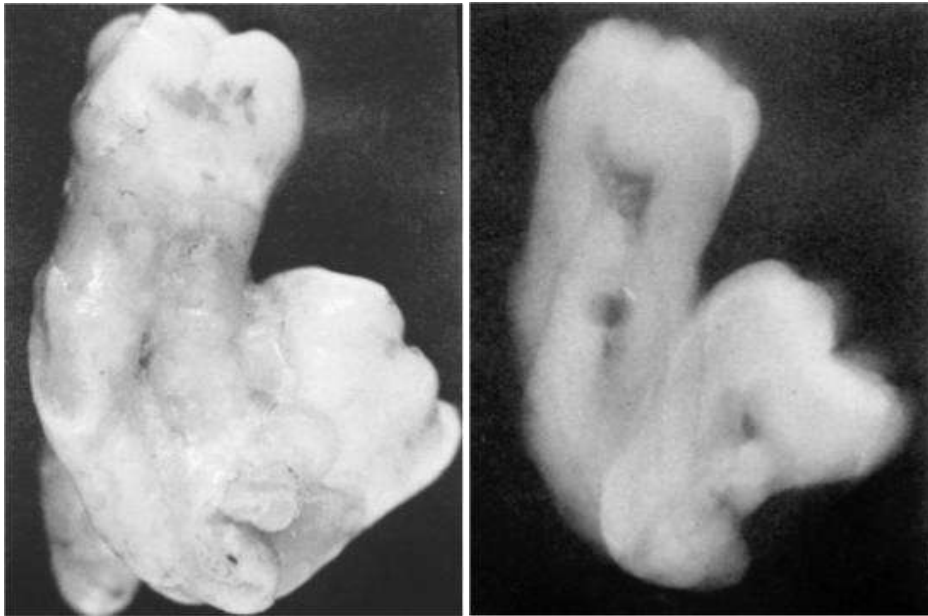
*Figure 62. Fusion.*

Tooth gemination is a dental phenomenon that appears to be two teeth developed from one. There is one main crown with a cleft in it that, within the cervical third of the crown, looks like two teeth, though it is not two teeth. The number of the teeth in the arch will be normal. The phenomenon of gemination arises when two teeth develop from one tooth bud and, as a result, the patient has an extra tooth, in contrast to fusion, where the patient would appear to be missing one tooth. Fused teeth arise through union of two normally separated tooth germs, and depending upon the stage of development of the teeth at the time of union, it may be either complete or incomplete. On some occasions, two independent pulp chambers and root canals can be seen. However, fusion can also be the union of a normal tooth bud to a supernumerary tooth germ. In these cases, the number of teeth is also normal and differentiation from gemination may be very difficult, if not impossible. In geminated teeth, division is usually incomplete and results in a large tooth crown that has a single root and a single canal. Both gemination and fusion are prevalent in primary dentition, with incisors being more affected.



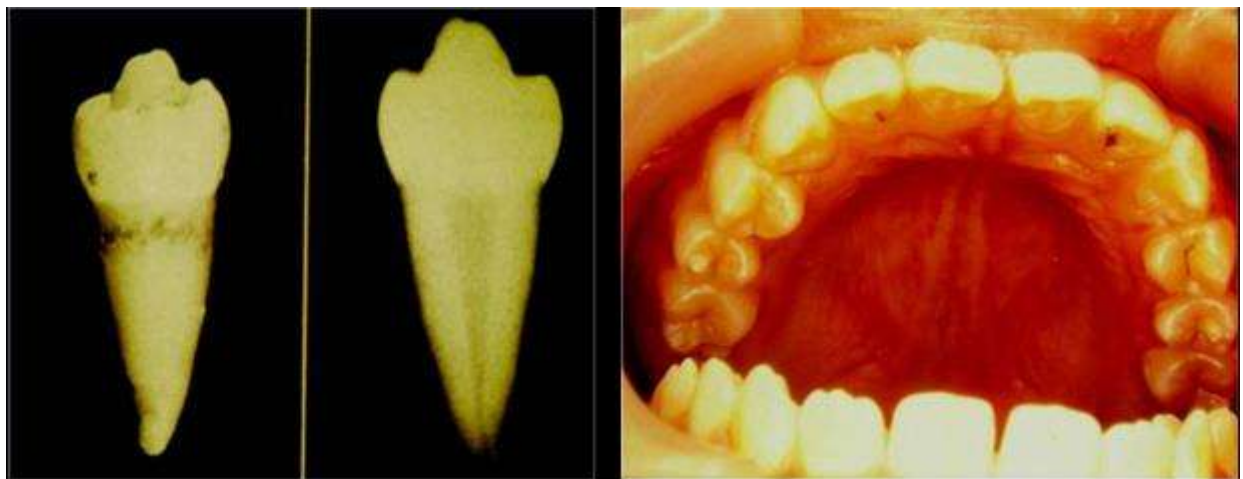
*Figure 63. Gemination.*

Concrescence is a condition of teeth where the cementum overlying the roots of at least two teeth join together. The cause can sometimes be attributed to trauma or crowding of teeth. Surgical separation of the teeth may be necessary if one is to be extracted.



*Figure 64. Concrescence.*

Dens evaginatus is a condition found in teeth where the outer surface appears to form an extra bump or cusp. Premolars are more likely to be affected than any other tooth. This may be seen more frequently in Asians, but almost exclusively in Down Syndrome. The pulp of the tooth may extend into the dens evaginatus. There is a risk of the dens evaginatus chipping off in normal function. Hence this condition requires monitoring as the tooth can lose its blood and nerve supply as a result and may need root canal treatment.



*Figure 65. Dens evaginatus.*

A talon cusp, also known as an "eagle's talon", is an extra cusp on an anterior tooth. The term refers to the same condition as dens evaginatus, but the talon cusp is the manifestation of dens evaginatus on anterior teeth. The incidence has been found to range from less than 1% to 6% of the population. Of all cases, 55% occur on the permanent maxillary lateral incisor, and 33% occur on the permanent maxillary central incisor. They are found rarely in primary teeth.

The condition is usually benign, but it can cause mild irritation to soft tissues around the teeth and the tongue, and if large enough, may pose an aesthetic problem. Talon cusps that are too large are filed down with a motorised file, and then endodontic therapy is administered.



*Figure 66. Talon cusp.*

Dens invaginatus, also known as dens in dente ("tooth within a tooth") is a condition found in teeth where the outer surface folds inward. There are coronal and radicular forms, with the coronal form being more common.

Dens invaginatus is a malformation of teeth most likely resulting from an infolding of the dental papilla during tooth development or invagination of all layer of the enamel organ in dental papillae. Affected teeth show a deep infolding of enamel and dentine starting from the foramen coecum or even the tip of the cusps and which may extend deep into the root. Teeth most affected are maxillary lateral incisors and bilateral occurrence is not uncommon. The malformation shows a broad spectrum of morphologic variations and frequently results in early pulp necrosis. Root canal therapy may present severe problems because of the complex anatomy of the teeth. Aetiology, prevalence, classification, and therapeutic considerations including root canal therapy, apical surgery and prevention of pulpal involvement are reviewed.



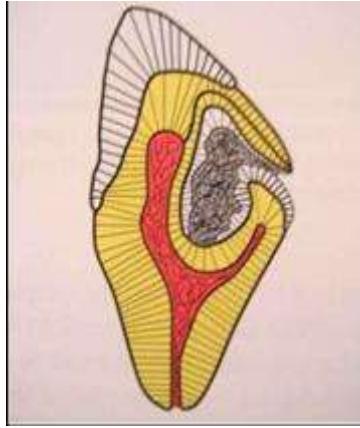
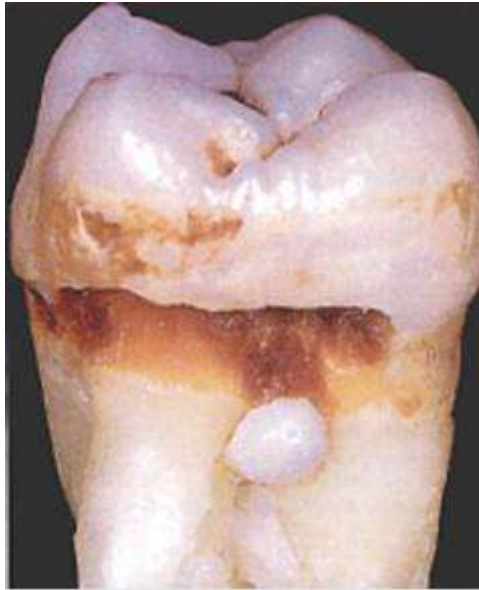


Figure 67. *Dens invaginatus*.

An enamel pearl is a condition of teeth where enamel is found on locations where enamel is not supposed to be, such as on a root surface. They are found usually in the area between roots, which is called a furcation, of molars. Enamel pearls are not common in teeth with a single root. The most common location of enamel pearl is the furcation areas of the maxillary and mandibular third molar roots. The Enamel pearls are formed essentially from the Hertwig's Epithelial root sheath. After the initiation of the formation of dentin in the root area of the tooth, the root sheath disintegrates and moves away from the root surface so that the cells of the dental sac can come in contact of predentin to differentiate into cementoblasts and start deposition of cementum. However, if the cells of epithelial root sheath remain adherent to predentin, they may differentiate into fully functional ameloblasts and deposit Enamel. Such droplets of enamel are called Enamel Pearls.

Macrodonia (or megadontia or megalodontia) is a type of localized gigantism in which teeth are larger than normal for the particular type(s) of teeth involved. The three types of macrodonia are true generalized macrodonia, relative generalized macrodonia, and macrodonia of a single tooth. True generalized macrodonia is rare. Macrodonia of a single tooth is more common. Some kind of macrodonia in the permanent dentition occurs in 1.1% of the total population. It should not be confused with taurodontism (bull teeth), fusion (double tooth) or the jaws being relatively small, giving the appearance of macrodonia. Males tend to have larger teeth than females, and tooth size also varies according race. Abnormal tooth size is defined by some as when the dimensions are more than 2 standard deviations from the average. Macrodonia is when the teeth are abnormally large, and microdonia when they are abnormally small. Macrodonia of a single tooth is attributed to a disturbance of morphodifferentiation. Generalized macrodonia is usually attributed to some hormonal imbalance (e.g., pituitary gigantism). It can also be associated with facial hemihyperplasia.



*Figure 68. Enamel pearl.*

True generalized microdontia. All the teeth are smaller than the normal size. True generalized microdontia is very rare, and occurs in pituitary dwarfism. Due to decreased levels of growth hormone the teeth fail to develop to a normal size.

Relative generalized microdontia. All the teeth are normal size but appear smaller relative to enlarged jaws. Relative generalized microdontia may be the result of inheritance of a large jaw from one parent, and normal sized teeth from the other.

Localized (focal) microdontia. Localized microdontia is also termed focal, or pseudo-microdontia. A single tooth is smaller than normal. Localized microdontia is far more common than generalized microdontia, and is often associated with hypodontia (reduced number of teeth). Females are affected more than males, and the condition occurs in permanent (adult) teeth more than deciduous (baby teeth or milk teeth). The most commonly involved tooth in localized microdontia is the maxillary lateral incisor, which may also be shaped like an inverted cone (a “peg lateral”). Peg laterals typically occur on both sides, and have short roots. Inheritance may be involved, and the frequency of microdontia in the upper laterals is just under 1%. The second most commonly involved tooth is the maxillary third molars, and after this supernumary teeth.



*Figure 69. Localized (focal) microdontia.*

There are many potential factors involved.

- ✓ Congenital hypopituitarism.
- ✓ Ectodermal dysplasia.
- ✓ Down syndrome.
- ✓ Ionizing radiation to the jaws during tooth development (odontogenesis).
- ✓ Chemotherapy during tooth development.
- ✓ Marshall syndrome.
- ✓ Rieger syndrome.
- ✓ Focal dermal hypoplasia.
- ✓ Silver-Russell syndrome.
- ✓ Williams syndrome.
- ✓ Gorlin-Chaudhry-Moss syndrome.
- ✓ Coffin–Siris syndrome.
- ✓ Salamon syndrome.
- ✓ Cleft lip and palate.

Others include trichorhinopharyngeal, odontotrichomelic, neuroectodermal and dermo-odontodysplasia syndromes.

Unerrupted microdonts may require surgical removal to prevent the formation of cysts. Erupted microdonts, peg laterals especially, may cause cosmetic concern. Such teeth may be restored to resemble normal sized teeth, typically with composite build ups or crowns. Orthodontics may be required in severe cases to close gaps between the teeth.

Taurodontism is a condition found in the molar teeth of humans whereby the body of the tooth and pulp chamber is enlarged vertically at the expense of the roots. As a result, the floor of the pulp and the furcation of the tooth is moved apically down the root. The underlying mechanism of taurodontism is the failure or late invagination of Hertwig's epithelial root sheath, which is responsible for root formation and shaping causing an apical shift of the root furcation.



The constriction at the amelocemental junction is usually reduced or absent. Taurodontism is most commonly found in permanent dentition although the term is traditionally applied to molar teeth. In some cases taurodontism seems to follow an autosomal dominant type of inheritance.

Taurodontism is found in association with amelogenesis imperfecta, ectodermal dysplasia and tricho-dento-osseous syndrome. The term means “bull like” teeth derived from similarity of these teeth to those of ungulate or cud-chewing animals.

It has also been reported in Klinefelter’s syndrome, XXYY and Down’s syndrome. The teeth involved are invariably molars, sometimes single and at the other times multiple teeth may be involved. The teeth themselves may look normal and do not have any particular anatomical character on clinical examination.

On a dental radiograph, the involved tooth looks rectangular in shape without apical taper. The pulp chamber is extremely large and the furcations may be only a few millimeters long at times.

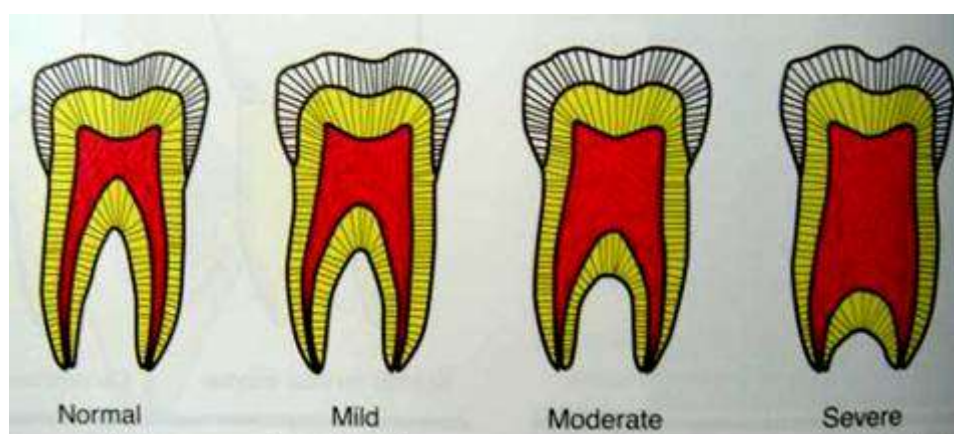


Figure 70. Taurodontism.

### Question 5. Mottled teeth.

**K00.3 Mottled teeth.** Dental fluorosis, also called mottling of tooth enamel, is a developmental disturbance of dental enamel caused by the consumption of excess fluoride during tooth development. Fluorosis continues to be an endemic problem. The following countries have been identified for the problem of fluorosis: Pakistan, Bangladesh, Argentina, United States of America, Morocco, Middle East countries, Japan, South African Countries, New Zealand, Thailand etc. Children in the age group of 0 to 12 years are most prone to fluorosis as their body tissues are in formative / growth stage during this period. Expectant mothers are also to be protected, as there is growing concern about effects of fluoride on fetus. Acceptable measures of fluorine in water according to hygienic standards is 0,8-1,2(1,5)mg/l.

Many well-known sources of fluoride may contribute to overexposure including dentifrice/fluoridated mouthrinse (which young children may swallow), bottled waters which are not tested for their fluoride content, inappropriate use of fluoride supplements, ingestion of foods especially imported from other countries, and public water fluoridation. The last of these sources is directly or indirectly responsible for 40% of all fluorosis, but the resulting effect due to water fluoridation is largely and typically aesthetic. Severe cases can be caused by exposure to water that is naturally fluoridated to levels well above the recommended levels, or by exposure to other fluoride sources such as brick tea or pollution from high fluoride coal.

Fluorosis is the term given to changes in the enamel which are associated with excess ingestion of fluoride. These vary from localised white opacities to more severe brown–yellow mottling on the teeth. The precise effect depends on the dose of fluoride (from all sources), the duration for which it was taken and the age of the patient at the time of ingestion. Fluorosis when very severe (concentrations in the water supply greater than six parts per million) may result in extensive hypoplasia with brown staining. Teeth are generally composed of hydroxyapatite and carbonated hydroxyapatite; as the intake of fluoride increases, so does the teeth's composition of fluorapatite. Excessive fluoride can cause white spots and, in severe cases, brown stains, pitting, or mottling of the enamel. A tooth is no longer at risk of fluorosis after eruption into the oral cavity. At this point, fluorapatite is beneficial because it is more resistant to dissolution by acids (demineralization). Although fluorosis usually affects permanent teeth, occasionally the primary teeth may be involved.

#### **Distinctive features of dental fluorosis**

- Mottling is endemic in areas where fluorides in the drinking water exceed about 2 parts per million, i.e. it has a geographical distribution.
- Neighbouring communities with fluoride-free water do not suffer from the disorder.
- Only those who have lived in a high-fluoride area during dental development show mottling. The defect is not acquired by older visitors to the area.
- Permanent teeth are affected; mottling of deciduous teeth is rare.
- Mottled teeth are less susceptible to caries than normal teeth from low-fluoride areas.
- A typical effect is paper-white enamel opacities.
- Brown staining of these patches may be acquired after eruption

**Dean's Index.** H.T. Dean's fluorosis index was first published in 1934. The index underwent two changes, appearing in its final form in 1942. This form became the most universally accepted classification system for dental

fluorosis. An individual's fluorosis score is based on the most severe form of fluorosis found on two or more teeth.

Table 49. **Dean's Index**

<b>Classification</b>	<b>Criteria – description of enamel</b>
Normal	Smooth, glossy, pale creamy-white translucent surface
Questionable	A few white flecks or white spots
Very Mild	Small opaque, paper white areas covering less than 25% of the tooth surface
Mild	Opaque white areas covering less than 50% of the tooth surface
Moderate	All tooth surfaces affected; marked wear on biting surfaces; brown stain may be present
Severe	All tooth surfaces affected; discrete or confluent pitting; brown stain present

**Clinical features.** Mottling ranges from paper-white patches to opaque, brown, pitted and brittle enamel. Clinically, it may be difficult to distinguish fluorotic defects from amelogenesis imperfect when the degree of exposure to fluoride is unknown. There is considerable individual variation in the effects of fluorides. A few patients acquire mottling after exposure to relatively low concentrations, while others exposed to higher concentrations appear unaffected.

#### **Grading of mottled enamel**



Figure 71. *Mottled enamel.*

- *Very mild.* Small paper-white areas involve less than 25% of surface.
- *Mild.* Opaque areas involve up to 50% of surface.
- *Moderate.* The whole of the enamel surface may be affected with paper-white or brownish areas or both.
- *Severe.* The enamel is grossly defective, opaque, pitted, stained brown and brittle.

**Treatment.** Dental fluorosis can be cosmetically treated by a dentist. The cost and success can vary significantly depending on the treatment. Tooth bleaching, microabrasion, and conservative composite restorations or porcelain veneers are commonly used treatments. Generally speaking, bleaching and microabrasion are used for superficial staining, whereas the conservative restorations are used for more unaesthetic situations.

#### **Question 6. Disturbances in tooth formation**

***K00.4 Disturbances in tooth formation.*** Dilaceration is a developmental disturbance in shape of teeth. It refers to an angulation, or a sharp bend or curve, in the root or crown of a formed tooth. The condition is thought to be due to trauma or possibly a delay in tooth eruption relative to bone remodeling gradients during the period in which tooth is forming. The result is that the position of the calcified portion of the tooth is changed and the remainder of the tooth is formed at an angle.

The curve or bend may occur anywhere along the length of the tooth, sometimes at the cervical portion, at other times midway along the root or even just at the apex of the root, depending upon the amount of root formed when the injury occurred.

Such an injury to a permanent tooth, resulting in dilaceration, often follows traumatic injury to the deciduous predecessor in which that tooth is driven apically into the jaw.



*Figure 72. Disturbances in tooth formation.*

Regional odontodysplasia or odontogenesis imperfecta is a developmental disturbance consisting of both enamel and dentin abnormalities in several adjacent teeth. The often-added suffix “regional” emphasises this usually localised character, but a few cases have been described with involvement of more extensive parts of the dentition, the abnormal teeth being present bilaterally and in both upper and lower jaw. The condition is nonhereditary. There is no predilection for race, but females are

more likely to get regional odontodysplasia. The enamel, dentin, and pulp of teeth are affected, to the extent that the affected teeth do not develop properly. These teeth are very brittle. The teeth are abnormally formed and the covering enamel layer is thin and yellow. The pulp chambers are wide and the amount of dentin is greatly reduced. The enamel is hypoplastic and the dentin contains large areas of interglobular dentin. Also, the predentin zone is very wide. The dental pulp usually contains large and irregular aggregates of mineralised matrix, the so-called denticles. The condition may be accompanied by gingival enlargement.

On radiographs the teeth appear more radiolucent than normal, so they are often described as "ghost teeth". Most cases are considered idiopathic, but some cases are associated with syndromes, growth abnormalities, neural disorders, and vascular malformations. Permanent teeth usually show effects of regional odontodysplasia if the deciduous tooth was affected. Many of these teeth do not erupt, and those that do have an increased risk of caries and periapical inflammation.



*Figure 73. Regional odontodysplasia.*

Treatment and prognosis are usually based upon keeping these teeth and devitalized and restorable. For unerupted teeth, function can be restored with a removable partial denture until all major growth has been completed and a final restoration can be placed.

### **Question 7. Enamel hypoplasia.**

Enamel hypoplasia is a defect of the teeth in which the enamel is hard but thin and deficient in amount, caused by defective enamel matrix formation with a deficiency in the cementing substance. Usually the condition involves part of the tooth having a pit in it. In some cases, the natural enamel crown has a hole in it, and in extreme cases, the tooth has no

enamel, which doesn't mean the tooth doesn't exist because dentin is also a component of teeth. It can be caused by any of the following:

- Malnutrition.
- Low birthweight.
- Prematurity.
- Maternal illness.
- Smoking.
- Drug abuse.
- Liver disease.
- Other systemic diseases.

If only a single tooth is affected these are classified as localized hypoplasia, but if several teeth are affected then they are referred to as generalized.

**Table50. Hypoplasia**

	<i>Generalized (systemic)</i>	<i>Localized</i>
Etiology	Premature birth, rhesus incompatibility, diseases suffered in childhood (from 0-9 months of life), intake of medicaments (antibiotics), malnutrition, lack of vitamins. Hypoplasia of temporary teeth: diseases of expecting mother, intake of toxic medicaments, malnutrition, smoking	Infection (follicles of premolars are placed between roots of temporary molars that are very often have apical periodontitis). Trauma to the deciduous predecessor (intrusive luxation of temporary tooth leads to injure of permanent follicle). These teeth are sometimes referred to as Turner teeth.
Affected teeth	Symmetrical teeth (those that have the same period of mineralization: maxilla central incisors and 1-st molars)	Permanent teeth, usually one tooth. The most common teeth affected due to trauma are anterior teeth; due to infection of predecessors are premolars.
Clinical manifestation	It is manifested in such forms: spotted, pitted, grooved, and linear. The more pronounced defect, the more severe was disease that caused it.	Single white (yellowish-brown) spot on incisal edge or in the equator of tooth (caries-immune zones). Turner tooth – enamel could be partly or totally absent, tooth could have pits, grooves, be brown-yellowish in color.
Objectively	Enamel is shine, hard and smooth on probing and not painful.	
Complaints of the patient	Aesthetic defect.	
Diagnosis	Diagnose is set from anamnesis of patient's life, anamnesis of disease and by applying extra methods of examination – vital coloring (methylene blue solution 2%) is negative.	
Differential diagnosis	Fluorosis, white spot lesion (initial caries)	Caries in the stage of white spot (initial caries)

Treatment and prophylaxis	Prophylaxis should be aimed at: care of general health of expectant mothers and later about newborns; prophylaxis of infectious and noninfectious diseases of kids in early childhood period; effective and timely treatment of somatic diseases (acute infectious diseases, malnutrition that leads to disease, hypo-, avitaminosis, etc.); increased awareness about this disease, is done as explanation to expectants mothers about proper lifestyle during pregnancy.	Prophylaxis: to prevent injuries of maxillofacial region in children; timely treatment of temporary teeth to prevent progression of infection in periapical region.
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Localised defects most commonly affect the upper incisor teeth or premolars and usually form as a result of infection (follicles of premolars are placed between roots of temporary molars that are very often have apical periodontitis) or trauma to the deciduous predecessor. This affects the ameloblasts of the developing tooth, resulting in the production of enamel which may be yellow–brown, pitted or irregular. These teeth are sometimes referred to as Turner teeth. Generalised defects are sometimes referred to as chronological hypoplasias and most are the result of a generalised or systemic infection or disturbance occurring during tooth development. The disturbance affects enamel formation and results in a linear horizontal band of hypoplasia. This may be characterised by ridging or grooving or pitting on the enamel surface and the teeth that are affected are those which were forming at the time of the disturbance. Hypoplastic defects that are localized on incisal edge of maxilla central incisors, canines and cusps of 1-st molars and lateral mandible incisors as well - could justify the disturbance in mineralisation at the age from 6 month until 1 year of life. Thus with a knowledge of the times of crown formation it is possible to predict at what age a patient was systemically unwell. The overwhelming majority of chronological hypoplasia as is the result of disturbances in the first 10 months of life and the teeth affected are:

- The first permanent molars.
- Upper central incisors.
- Lower lateral incisors and canines.

**Table 51. Timing of eruption and calcification**

	<i>Calcification begins (months)</i>	<i>Eruption (years)</i>
<b><i>Upper:</i></b>		
Central incisor	3-4	7-8



Lateral incisor	10-12	8-9
Canine	4-5	11-12
First premolar	18-21	10-11
Second premolar	24-27	10-12
First molar	At birth	6-7
Second molar	30-36	12-13
Third molar	84-108	17-21
<b><i>Lower:</i></b>		
Central incisor	3-4	6-7
Lateral incisor	3-4	7-8
Canine	4-5	9-10
First premolar	21-24	10-12
Second premolar	27-30	11-12
First molar	At birth	6-6
Second molar	30-36	12-13
Third molar	96-120	17-21
Root calcification is complete 2-3 years after eruption.		
Typical eruption sequence: upper 6 1 2 4 5 3 7 8; lower 6 1 2 3 4 5 7 8.		

Turner's hypoplasia is an abnormality found in teeth. Its appearance is variable, though usually is manifested as a portion of missing or diminished enamel on permanent teeth. Unlike other abnormalities which affect a vast number of teeth, Turner's hypoplasia usually affects only one tooth in the mouth and, it is referred to as a Turner's tooth.



*Figure 74. Turner's hypoplasia.*

If Turner's hypoplasia is found on a canine or a premolar, the most likely cause is an infection that was present when the primary (baby) tooth



was still in the mouth. Most likely, the primary tooth was heavily decayed and an area of inflamed tissues around the root of the tooth (called a periapical inflammation), affecting the development of the permanent tooth. The tooth most likely affected by this cause is the canine tooth. The appearance of the abnormality will depend on the severity and longevity of the infection.

If Turner's hypoplasia is found in the front (anterior) area of the mouth, the most likely cause is a traumatic injury to a primary tooth. The traumatized tooth, which is usually a maxillary central incisor, is pushed into the developing tooth underneath it and consequently affects the formation of enamel. Because of the location of the permanent tooth's developing tooth bud in relation to the primary tooth, the most likely affected area on the permanent tooth is the facial surface (the side closer to the lips or cheek). White or yellow discoloration may accompany Turner's hypoplasia. Enamel hypoplasia may also be present.

Turner's hypoplasia usually affects the tooth enamel if the trauma occurs prior to the third year of life. Injuries occurring after this time are less likely to cause enamel defects since the enamel is already calcified.

One of the forms of systemic hypoplasia is Hutchinson's teeth. Prenatal syphilis, the result of maternal infection, can cause a characteristic dental deformity, described by Hutchinson in 1858. If the fetus becomes infected at a very early stage, abortion follows. Infants born with stigmata of congenital syphilis result from later fetal infection, and the permanent teeth are affected. The characteristic defects are usually seen in the upper central incisors.



*Figure 75. Hutchinson's incisors.*

The incisors (Hutchinson's incisors) are small, barrel-shaped, and taper towards the tip. The incisal edge sometimes shows a crescentic notch or deep fissure which forms before eruption and can be seen radiographically. An anterior open bite is also characteristic. The first molars may be dome-shaped

(Moon's molars) or may have a rough pitted occlusal surface with compressed nodules instead of cusps (mulberry molars). These defects are now largely of historical interest.

**Pathology.** The effects are due to infection of the dental follicle by *Treponema pallidum*. The postulated consequences are chronic inflammation, fibrosis of the tooth sac, compression of the developing tooth and distortion of the ameloblast layer. *T. pallidum* causes proliferation of the odontogenic epithelium which bulges into the dentine papilla causing the characteristic central notch.

### **Question 8. Tetracycline discoloration.**

Tetracycline is taken up by calcifying tissues, and the band of tetracycline-stained bone or tooth substance fluoresces bright yellow under ultraviolet light. The teeth become stained only when tetracycline is given during their development, and it can cross the placenta to stain the developing teeth of the fetus. More frequently, permanent teeth are stained by tetracycline given during infancy. Tetracycline is deposited along the incremental lines of the dentine and, to lesser extent, of the enamel. The more prolonged the course of treatment the broader the band of stain and the deeper the discolouration. The teeth are at first bright yellow, but become a dirty brown or grey. The stain is permanent, and when the permanent incisors are affected the ugly appearance can only be disguised. When the history is vague the brownish colour of tetracycline-stained teeth must be distinguished from dentinogenesis imperfecta. In dentinogenesis imperfecta the teeth are obviously more translucent than normal and, in many cases, chipping of the enamel from the dentine can be seen. In tetracycline-induced defects the enamel is not abnormally translucent and is firmly attached to dentine. In very severe cases, intact teeth may fluoresce under ultraviolet light. It is no longer necessary to give tetracycline during dental development. There are equally effective alternatives and it should be avoided from approximately the fourth month to 12th year of childhood. Nevertheless tetracycline pigmentation is still seen.



Figure 76. Tetracycline discoloration.

Trough binding to calcium, tetracyclin is deposited together with calcium in any tissue undergoing mineralisation. After its incorporation during mineralisation, it can be demonstrated in teeth and bones in ultraviolet light, showing up as fluorescent yellow bands. Grossly, tetracyclin causes a grey-black discoloration of the tooth crown.



Figure 77. Tetracycline discoloration.

When making ground sections of these teeth, the tetracyclin bands can be observed under UV light illumination both in dentin as well as in enamel, each band indicating a time point of tetracyclin administration. After decalcification the tetracyclin has been lost together with the calcium and therefore, in decalcified paraffin sections, this fluorescence is not present anymore.

**Treatment of hypoplastic defects.** Hypoplastic teeth can be disguised by restorative procedures such as veneers or jacket crowns. The latter should be delayed until adult life. The young pulp is large, is easily damaged during preparation of the tooth, and injuries are more frequent than in older persons.

**Localised composite resin restorations.** Defective enamel can be replaced with a tooth-coloured restoration that bonds to, and blends with, enamel. It is indicated for well-demarcated white, yellow or brown patches. The localised restoration is quick and easy to complete. Despite the removal of defective enamel down to the amelodentine junction, there is often no significant sensitivity and, therefore, no need for local anaesthesia. If the hypoplastic enamel has become carious and extends into dentine, a liner of glass ionomer cement (correct shade) prior to placement of composite resin will be necessary. In these cases, local anaesthesia will probably be required. Advances in bonding and resin technology make these restorations simple and obviate the need for a full labial veneer. Disadvantages are marginal staining, difficulty in achieving an accurate colour match and reduced composite translucency when lined by a glass ionomer cement.

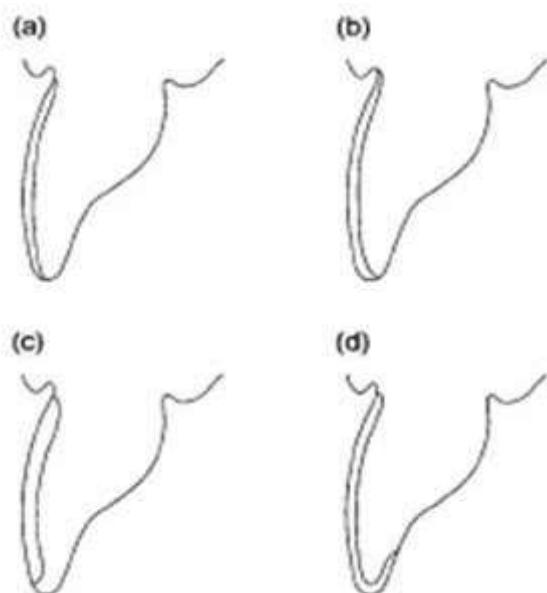
**Composite resin veneers.** Although the porcelain jacket crown (PJC) may be the most satisfactory long-term restoration for a severely hypoplastic or discoloured tooth, it is not an appropriate solution for children for two reasons: the large size of the young pulp horns and chamber, and the immature gingival contour. Composite veneers may be direct (placed at initial appointment) or indirect (placed at a subsequent appointment having been fabricated in the laboratory). The conservative veneering methods may not just offer a temporary solution but may also offer a satisfactory long-term alternative to the PJC. Most composite veneers placed in children and adolescents are of the 'direct' type as the durability of the indirect composite veneers is as yet unknown. Composite veneers are durable enough to last through adolescence. Before proceeding with any veneering technique, the decision must be made whether to reduce the thickness of labial enamel before placing the veneer. Certain factors should be considered:

- increased labiopalatal bulk makes it harder to maintain good oral hygiene; this may be courting disaster in the adolescent with dubious oral hygiene
- composite resin has a better bond strength to enamel when the surface layer of 200-300 µm is removed
- if a tooth is very discoloured, some sort of reduction will be desirable as a thicker layer of composite will be required to mask the intense stain
- if a tooth is already instanding or rotated, its appearance can be enhanced by a thicker labial veneer.

New-generation, highly polishable hybrid composite resins can replace relatively large amounts of missing tooth tissue as well as being used in thin sections as a veneer. Combinations of shades can be used to stimulate natural colour gradations and hues. The exact design of the composite veneer will vary with each patient. Usually it will be one of four types: intraenamel or window preparation, incisal bevel, overlapped incisal edge or feathered incisal edge.

**Indications:**

- ✓ discoloration,
- ✓ enamel defects,
- ✓ diastemata,
- ✓ malpositioned teeth,
- ✓ large restorations.



*Figure 78. Types of veneer preparation*  
(a) Feathered incisal edge, (b) Incisal bevel preparation,  
(c) Intra-enamel or window preparation,  
(d) Overlapped incisal edge preparation.

**Question 9. Hereditary disturbances in tooth structure,  
not elsewhere classified.**

***K00.5 Hereditary disturbances in tooth structure, not elsewhere classified***

Amelogenesis imperfecta (AI) presents with a rare abnormal formation of the enamel or external layer of the crown of teeth. Enamel is composed mostly of mineral, that is formed and regulated by the proteins in it. Amelogenesis imperfecta is due to the malfunction of the proteins in the enamel: ameloblastin, enamelin, tuftelin and amelogenin.

Mutations in the AMELX, ENAM, MMP20, KLK-4, FAM83H, WDR72, C4orf26, SLC24A4 LAMB3 and ITGB6 genes have been found to cause amelogenesis imperfecta (non-syndromic form). AMELX and ENAM encode extracellular matrix proteins of the developing tooth enamel and KLK-4 and MMP20 encode proteases that help degrade organic matter from the enamel matrix during the maturation stage of amelogenesis. SLC24A4 encodes a calcium transporter that mediates calcium transport to developing

enamel during tooth development. Less is known about the function of other genes implicated in amelogenesis imperfecta.

Amelogenesis imperfecta can have different inheritance patterns depending on the gene that is altered. Mutations in the ENAM gene are the most frequent known cause and are most commonly inherited in an autosomal dominant pattern. This type of inheritance means one copy of the altered gene in each cell is sufficient to cause the disorder.

Amelogenesis imperfecta is also inherited in an autosomal recessive pattern; this form of the disorder can result from mutations in the ENAM, MMP20, KLK4, FAM20A, C4orf26 or SLC24A4 genes. Autosomal recessive inheritance means two copies of the gene in each cell are altered.

About 5% of amelogenesis imperfecta cases are caused by mutations in the AMELX gene and are inherited in an X-linked pattern. A condition is considered X-linked if the mutated gene that causes the disorder is located on the X chromosome, one of the two sex chromosomes. In most cases, males with an X-linked form of this condition experience more severe dental abnormalities than affected females.

Recent genetic studies suggest that the cause of a significant proportion of amelogenesis imperfecta cases remains to be discovered.

People afflicted with amelogenesis imperfecta have teeth with abnormal color: yellow, brown or grey; this disorder can afflict any number of teeth of both dentitions. The teeth have a higher risk for dental cavities and are hypersensitive to temperature changes as well as rapid attrition, excessive calculus deposition, and gingival hyperplasia.

The clinical features are variable and depend on which gene is defective:

- ✓ In some forms the teeth appear normal on eruption but, because the enamel is poorly mineralised, it is soft and soon wears away. The remaining enamel becomes stained and the teeth appear yellow–brown.
- ✓ In other forms the enamel is reduced in amount and the teeth are pitted and appear yellow–brown in colour.
- ✓ Some forms are carried on the X chromosome and boys are affected more severely than girls.
- ✓ The teeth are often sensitive, especially where the enamel is thin or has chipped away.

Hypomaturation amelogenesis imperfecta. The enamel is normal in form on eruption but opaque, white to brownish-yellow. The teeth appear similar to mottled fluoride effects. However, they are soft and vulnerable to attrition, though not as severely as the hypocalcified type. There are several variants of hypomaturation defects such as a more severe, autosomal dominant (type 4) of hypomaturation combined with hypoplasia.



Figure 79. Amelogenesis imperfecta, hypomaturational type.

Hypocalcified amelogenesis imperfecta. Enamel matrix is formed in normal quantity but poorly calcified. When newly erupted, the enamel is normal in thickness and form, but weak and opaque or chalky in appearance. The teeth tend to become stained and relatively rapidly worn away. The upper incisors may acquire a shouldered form due to the chipping away of the thin, soft enamel of the incisal edge. There are dominant and recessive patterns of inheritance.



Figure 80. Amelogenesis imperfecta, hypocalcified type.

**Treatment.** Preventive and restorative dental care is very important as well as considerations for esthetic issues since the crown are yellow from exposure of dentin due to enamel loss. Full-coverage crowns are sometimes being used to compensate for the abraded enamel. Usually stainless steel crowns are used in children which may be replaced by porcelain once they reach adulthood. In the worst-case scenario, the teeth may have to be extracted and implants or dentures are required. Loss of nerves in the affected teeth may occur.

Dentinogenesis imperfecta is a genetic disorder of tooth development. This condition is a type of dentin dysplasia that causes teeth to be discolored



(most often a blue-gray or yellow-brown color) and translucent giving teeth an opalescent sheen. Teeth are also weaker than normal, making them prone to rapid wear, breakage, and loss. These problems can affect both primary (deciduous) teeth and permanent teeth. This condition is inherited in an autosomal dominant pattern, which means one copy of the altered gene in each cell is sufficient to cause the disorder. Dentinogenesis imperfecta affects an estimated 1 in 6,000 to 8,000 people.



Figure 81. *Dentinogenesis imperfecta*

### ***Types of dentinogenesis imperfecta***

*Type I:* Type of dentinogenesis imperfecta with similar dental abnormalities usually an autosomal dominant trait with variable expressivity but can be recessive if the associated osteogenesis imperfecta is of recessive type.

*Type II:* Occurs in people without other inherited disorders (i.e. Osteogenesis imperfecta). It is an autosomal dominant trait. A few families with type II have progressive hearing loss in addition to dental abnormalities. Also called hereditary opalescent dentin.

*Type III:* Type is rare; its predominant characteristic is bell-shaped crowns, especially in the permanent dentition. Unlike Types I and II, it involves teeth with shell-like appearance and multiple pulp exposures.

Mutations in the DSPP gene have been identified in people with type II and type III dentinogenesis imperfecta. Type I occurs as part of osteogenesis imperfecta.

Clinical appearance is variable. However, the teeth usually involved and more severely affected are primary teeth in type I; whereas in type II both the dentitions are equally affected.

The teeth may be gray to yellowish brown. They exhibit translucent or opalescent hue. Enamel is usually lost early due to loss of scalloping at the



dentoenamel junction (DEJ). However, the teeth are not more susceptible to dental caries than normal ones.

However, certain patients with dentinogenesis imperfecta will suffer from multiple periapical abscesses apparently resulting from pulpal strangulation secondary to pulpal obliteration or from pulp exposure due to extensive coronal wear. They may need apical surgery to save the involved teeth.

**Radiographic features.** Type I and II show total obliteration of the pulp chamber. Type III shows thin dentin and extremely enormous pulp chamber. These teeth are usually known as "shell teeth".

**Histology.** Dentinal tubules are irregular and are bigger in diameter. Areas of uncalcified matrix are seen. Sometimes odontoblasts are seen in dentin.

**Treatment.** Preventive and restorative care are important as well as esthetics as a consideration. In most cases, full-coverage crowns are needed for esthetic appearance, as well as to prevent further attrition. Another treatment option is bonding, putting lighter enamel on the weakened enamel of the teeth and with lots of treatments of this bonding, the teeth appear whiter to the eye, but the teeth on the inside and under that cover are still the same. Due to the weakened condition of the teeth, many common cosmetic procedures such as braces and bridges are inappropriate for patients with Dentinogenesis imperfecta and are likely to cause even more damage than the situation they were intended to correct.

Dental whitening (bleaching) is contraindicated although it has been reported to lighten the color of DI teeth with some success; however, because the discoloration is caused primarily by the underlying yellow-brown dentin, this alone is unlikely to produce normal appearance in cases of significant discoloration.

Dentin dysplasia is a genetic disorder of teeth, commonly exhibiting an autosomal dominant inheritance. It is characterized by presence of normal enamel but atypical dentin with abnormal pulpal morphology. There are two types. Type I is the radicular type, and type II is the coronal type. In the radicular type, the roots of teeth are shorter than normal and the pulp chamber may be nearly gone. The pulp chamber is sometimes described as having a "crescent shaped" appearance. In the coronal type, the pulps are enlarged and are described as having a "thistle tube" appearance, in permanent dentition. In the deciduous dentition, coronal dentin dysplasia bears a resemblance to Dentinogenesis Imperfecta type II.

### Radiographic Features:

*Type 1:* Roots are short, blunt and conical. In deciduous teeth, pulp chambers and root canals are completely obliterated while in permanent they may be crescent shaped.

*Type 2:* The pulp chamber of the deciduous teeth become completely obliterated. The permanent teeth displays large pulp chamber in the coronal portion of the tooth referred to as thistle tube appearance. Pulp stones may be found.

### **Question10. Disturbances in tooth eruption.**

***K00.6 Disturbances in tooth eruption.*** Natal teeth are teeth that are present above the gumline (have already erupted) at birth, and neonatal teeth are teeth that emerge through the gingiva during the first month of life (the neonatal period).

- ✓ The incidence of neonatal teeth varies considerably, between 1:700 and 1:30,000 depending on the type of study; the highest prevalence is found in the only study that relies on personal examination of patients.
- ✓ Most often natal teeth are mandibular central incisors. They have little root structure and are attached to the end of the gum by soft tissue and are often mobile.
- ✓ Cause: a developmental disturbance creating intracellular activity during the first stage of tooth development (bud stage) can result in the development of extra teeth.
- ✓ Most of the time, natal teeth are not related to a medical condition. However, sometimes they may be associated with:
- ✓ Ellis–van Creveld syndrome.
- ✓ Hallermann–Streiff syndrome.
- ✓ Pierre Robin syndrome.
- ✓ Sotos syndrome.

***Treatment.*** Natal teeth: these teeth are defective and their removal is generally recommended, particularly if mobility poses a threat of aspiration. These teeth also make feeding difficult.

Neonatal teeth: these teeth are defective and their removal is generally recommended, particularly if mobility poses a threat of aspiration. These teeth also make feeding difficult.

### **Question 11. Classification of noncarious lesions that appear after teeth eruption.**

Noncarious tooth lesions is a common problem. After tooth eruption exposed dentin can result from acidic erosion, abrasion, and attrition, but most toothwear has erosion as the dominant etiological factor. Localized anterior toothwear of the upper anterior teeth is often caused by the

consumption of erosive carbonated beverages, fruit juices, and citrus fruits. Regurgitated stomach acid in gastroesophageal reflux disease, hiatus hernia, and esophagitis and vomiting in bulimia, alcoholism, and psychosomatic disorders can cause erosive tooth wear of the palatal surfaces of the anterior teeth. Drugs that tend to reduce the amount of saliva in the mouth, such as antidepressants, recreational drugs (LSD and Ecstasy, which is 3,4-methylene-dioxymethamphetamine), and diuretics, also diminish the buffering capacity available to neutralize dietary or stomach acids. Users of Ecstasy commonly complain of a dry mouth, and erosion from carbonated beverages is thought to be an important etiological factor. However, the occlusal surfaces of the molar teeth are more commonly affected than the incisor teeth, which would indicate that jaw clenching and masseter muscle hyperactivity are important (Milosevic et al. 1999).

According to International classification of diseases there are following noncarious lesions that appear after teeth eruption:

***K03 Other diseases of hard tissues of teeth***

***K03.0 Excessive attrition of teeth:***

- approximal
- occlusal

***K03.1 Abrasion of teeth:***

- dentifrice
- habitual
- occupational
- ritual
- traditional

Wedge defect

***K03.2 Erosion of teeth***

Erosion of teeth due to:

- diet
- drugs and medicaments
- persistent vomiting
- idiopathic
- occupational

***K03.3 Pathological resorption of teeth***

Internal granuloma of pulp

Resorption of teeth (external)

***K03.4 Hypercementosis***

Cementation hyperplasia

***K03.5 Ankylosis of teeth***

***K03.7 Posteruptive colour changes of dental hard tissues***

***K03.8 Other specified diseases of hard tissues of teeth***

Irradiated enamel

Sensitive dentine

### ***K03.9 Disease of hard tissues of teeth, unspecified***

#### **Question 12. Excessive attrition of teeth.**

***K03.0 Excessive attrition of teeth.*** Attrition is the process of wearing away of enamel, which occurs physiologically as a consequence of mastication. This process is very slow and results in a gradual loss of enamel. Pindborg distinguished three types of attrition: physiological, excessive and pathological. The dental pulp cavity is usually not open due to attrition. Dental attrition is a type of tooth wear caused by tooth-to-tooth contact, resulting in loss of tooth tissue, usually starting at the incisal or occlusal surfaces.

**Causes.** Advanced and excessive wear and tooth surface loss can be defined as pathological in nature, requiring intervention by a dental practitioner. Excessive occlusal wear may occur in the following situations:

- Patients with bruxism. These patients grind their teeth excessively and in some cases this is triggered by occlusal irregularities.
- Patients who have lost several posterior teeth may show excessive attrition of the anterior teeth, especially if these are used for chewing.
- Patients who suffer from developmental disturbances of tooth structure, such as amelogenesis and dentinogenesis imperfecta, may suffer exceptional tooth wear.

The etiology of dental attrition is multifactorial however bruxism is one of the most common causes of attrition. Bruxism is the parafunctional movement of the mandible, occurring during the day or night. It can be associated with presence of audible sound when clenching or grinding the teeth. This is usually reported by parents or partners if the grinding occurs during sleep.

In some cases dental erosion is also associated with severe dental attrition. Dental erosion is tooth surface loss caused by extrinsic or intrinsic forms of acid. Extrinsic erosion is due to a highly acidic diet, whilst intrinsic erosion is caused by regurgitation of gastric acids. Erosion softens the dental hard tissues making them more susceptible to dental attrition. When dental erosion is present in conjunction with bruxism the tooth surface loss due to attrition is accelerated due to the erosive environment. Severe attrition in young patients is usually associated with erosive factors in their diets. The different physiological processes of tooth wear (abrasion, attrition and erosion) generally occur simultaneously and rarely work individually. Therefore it is important to obtain knowledge of these tooth wear processes and their interactions to determine causes of tooth surface loss. Demineralization of the tooth surface due to acids can cause occlusal erosion

as well as attrition. Wedge-shaped cervical lesions are commonly found in association with occlusal erosion and attrition.

Tooth wear is typically seen in the elderly and can be referred to as a natural aging process. Attrition, abrasion, erosion or a combination of these factors are the main reasons for tooth wear in elderly people who retain their natural teeth. This tooth wear can be pathological or physiological in nature. The influence of age on tooth wear shows that the number of teeth with incisal or occlusal wear increases with the age. Dental attrition occurs in 1 in 3 adolescents and an association has been established between dental attrition and aging.

Gender has also been determined as a contributing factor associated with occlusal tooth wear. In addition to other occlusal factors some independent variables such as male gender, bruxism, and loss of molar occlusal contact, edge-to-edge relation of incisors, unilateral buccolingual cusp-to-cusp relation, and unemployment have been identified in affecting occlusal wear. Similarly anterior cross-bite, unilateral posterior cross-bite, and anterior crowding have been found to be protective factors for high occlusal wear levels.

Clinical indications of attrition can include:

- Loss of tooth anatomy: This results in loss of tooth characteristics including rounding or sharpening of incisal edges, loss of cusps and fracturing of teeth. Enamel of molar teeth may appear thin and flat. When in occlusion the teeth may appear the same height which is particularly apparent for anterior teeth.
- Sensitivity or pain: Attrition may be entirely asymptomatic, or there may be dentin hypersensitivity secondary to loss of the enamel layer, or tenderness of the periodontal ligament caused by occlusal trauma.
- Tooth discolouration: A yellow appearance of the tooth surface may be due to the enamel being worn away, exposing the darker yellower dentin layer underneath.
- Altered occlusion due to decreasing vertical height, or occlusal vertical dimension.
- Compromised periodontal support can result in tooth mobility and drifting of teeth.
- Loss in posterior occlusal stability.
- Mechanical failure of restorations.



*Figure 82. Excessive attrition of teeth.*

**Prevention and management.** To manage the condition it is first important to arrive at a diagnosis, describing the type of tooth surface loss, the severity and location. Early diagnosis is essential to ensure tooth wear has not progressed past the point of restoration. The examination should include assessment of

- ✓ Temporomandibular joint function and associated musculature
- ✓ Orthodontic examination
- ✓ Intraoral soft tissue analysis
- ✓ Hard tissue analysis
- ✓ Location and severity of tooth wear
- ✓ Social history;
- ✓ Diet.

It is important to record severity of tooth wear for monitoring purposes, helping to differentiate between pathological and physiological tooth surface loss. It is essential to determine whether the tooth wear is ongoing or has stabilized. However where generalised one can assume the underlying cause is bruxism. In fast progressing cases there is commonly a coexisting erosive diet contributing to tooth surface loss.

When a diagnosis of bruxism has been confirmed it is recommended that the patient purchase a full coverage acrylic occlusal splint. Patients must be monitored closely, with clinical photographs 6–12 monthly to evaluate if the tooth surface loss is being prevented.



*Figure 83. Occlusal splint.*

**Treatment.** Cosmetic or functional intervention may be required if tooth surface loss is pathological in nature or if there has been advanced loss of tooth structure. The first stage of treatment involves the management of any associated conditions such as fractured teeth or sharp cusps or incisal edges. These can be resolved via restoration of and polishing of sharp cusps. At this stage desensitizing agents such as topical fluoride varnishes can be applied, and at home desensitising toothpastes recommended. There are many different restorative treatment options which have been proposed such as direct composite restorations, bonded cast metal restorations, removable partial dentures, orthodontic treatment, crown lengthening procedures and protective splints. The decision to restore the dentition depends on the wants and needs of the patient, the severity of tooth surface loss and whether tooth surface loss is active. The use of adhesive materials to replace lost tooth structure can be performed as a conservative and cost effective approach before a more permanent solution of crowns or veneers is considered.

### **Question 13. Teeth abrasion.**

***K03.1 Abrasion of teeth.*** Abrasion is the loss of tooth structure by mechanical forces from a foreign element. If this force begins at the cemento-enamel junction, then progression of tooth loss can be rapid since enamel is very thin in this region of the tooth. Once past the enamel, abrasion quickly destroys the softer dentin and cementum structures.

Abrasion is seen at a cervical necks of the teeth, as a deep ridge on the buccal or labial surfaces. The surface is shiny rather than carious, and sometimes the ridge is deep enough to see the pulp chamber within the tooth itself. The teeth most commonly affected are premolars and canines.

#### **Causes of abrasion :**

- Traumatic occlusion.
- Unproper brushing technique.
- Occupational (Habits such as holding bobby pins in between the teeth).
- Tobacco chewing /tobacco pipe.
- Vigorous use of tooth picks between the adjacent teeth.
- Excessive mastication of coarse foods.

#### **Iatrogenic causes:**

- Dentures with porcelain teeth opposing natural teeth.
- Extremely rough occluding surface of the restoration enhancing its abrasive capability .
- Ill fitting dentures and clasps, producing a constant wear of the affected surfaces.



*Figure 84. Teeth abrasion.*

Tooth brush abrasion results in a horizontal cervical notches on the buccal surfaces of exposed radicular cementum and dentin. Notching in right central incisor caused by improper use of bobby pins.

The clinical signs and symptoms of an abrasion are:

- The surface of the lesion is extremely smooth and polished and it seldom has any plaque accumulation or caries activity in it.
- The surrounding walls tend to make a V shape ,by meeting at an acute angle axially.
- Peripheries of the lesion are angularly demarcated from the adjacent tooth surface.
- Probing or stimulating the lesion can elicit pain.
- Hypersensitivity may be intermittent in character appearing and disappearing at occasional or frequently repeated periods

#### **Treatment modalities**

1. Diagnose the cause of the presented abrasion.
2. A detailed history is to be taken considering various factors such as:
  - Oral hygiene techniques ( use of abrasive tooth cleaning techniques and materials)
  - Habits- pipe smoking, chewing tobacco, professional habits
  - Iatrogenic causes, if any.
3. Avoidance or counteraction of the causes which may lead to its production.
4. Instituting proper oral hygiene measures.
5. Judiciously tooth brushing with a dentifrice i.e. incorporating correct method of tooth brushing Have the habit of chewing tobacco ,toothpick , etc discontinued. If successful in breaking the habit proceed with the restorative treatment as planned.
6. Correcting or avoiding ill fitting metal clasps and dentures
7. Abrasive lesions at non-occluding tooth surfaces should be:
8. Evaluated critically for the need for restoring them.



9. If the lesions are multiple, shallow( not exceeding 0.5 mm in dentin) and wide → no need to restore them.

10. If there is involvement of cementum/enamel only → no need to restore .

11. If lesion is wedge (V) shaped and exceeds 0.5 mm into dentin → restoration is performed.

12. If restoration is not indicated for a lesion, then :

- edges of the defect should be eradicated to a smooth, non- demarcating pattern relative to adjacent tooth surface.
- tooth surface then should be treated by fluoride solution to improve caries resistance

13. If the involved teeth→ extremely sensitive: desensitize the exposed dentin before restoration.

**Desensitization:**

- 8-10% sodium/stannous fluorides for 4-8 minutes.
- Ionophoresis- -using an electrolyte containing fluorides( galvanic energy supplied to the tooth in the presence of electrolyte, drives ions deep into the dentin)

**Restorative materials.** High modulus restorative materials are unable to flex in the cervical regions when the tooth structure is deformed under occlusal load and ,therefore the restorative materials can be displaced from the cavity.

An intermediate material with reduced elastic modulus may function as a stress absorbing layer and improve marginal sealing.

As a result materials with low elastic modulus such as: microfilled composites, flowable resins, glass ionomer cements have been used in restoring cervical lesions ,with the aim of absorbing the stresses generated during the polymerization shrinkage of composites and mechanical loading in which the teeth are subjected during function.

**Question 14. Wedge defect.**

The wedge-shaped defects refers to the teeth, lips, buccal neck dental hard tissue slowly consumed due to defects. This defect was "shaped, like woodworking wedge, hence the name wedge-shaped defect. One at the junction of the wedge-shaped defects occur in the lip and cheek surfaces of the canine and premolar tooth enamel, some patients confined to unilateral disease some bilateral occurred, individual full mouth of teeth can occur. Have a greater difference in the extent of the defect in the same patient. Early there is a small amount of hard tissue defect gradually increased after the formation of tooth neck.

Defects at the tooth surface smooth, hard and without significant color change. Mild wedge-shaped defects may be no symptoms, heavy sensitive symptoms due to dentin exposure, performance eating a certain food, fruit or

brushing pain symptoms, defects gradually increased, a series of lesions can be caused by the pulp, or even because of defect caused by excessive teeth broken from the neck. The real cause of the wedge-shaped defect is unclear, may be related to the following factors.



*Figure 85. Wedge defect.*

Acid secretion. Chemical factors gingival sulcus often acting on the tooth neck, low pH of saliva, like to eat acidic foods, food residue accumulation of fermentation acid may cause dental hard tissue decalcification gradually dissolved and loss.

Some physical factors teeth mechanical friction often can accelerate dental hard tissue loss. Clinically found a higher incidence of horizontal brush teeth, lip and cheek surfaces using a hard bristle toothbrush, wedge-shaped defects.

#### **Treatments of Wedge-Shaped Defects**

Wedge-shaped defects early hard tissue defects and no obvious symptoms, local do not need special treatment, but to be paid to the choice of a soft bristle toothbrush with a the vertical brush or rotary brushing, and attention to weak alkaline gargle mouthwash: formation defect and allergic symptoms, do desensitization treatment or filling to repair the defect. Commonly desensitization many ways. Here are some kinds.

**1. Fluoride treatment, wedge-shaped defects.** Teeth topcoat fluorine may be formed - fluoride hydroxyapatite processing dentin hypersensitivity, fluoride ions can reduce the diameter of dentinal tubules and the formation of the fluorine-containing compound blocking the transmission of a small tube with the blocking stimulation, but also promote a more rigid dentin formation. Sodium fluoride treatment of wedge-shaped defects: 33% sodium fluoride paste is the most effective, with 75% sodium fluoride glycerin paste domestic. General use cotton balls dipped in paste repeatedly rubbed sensitive at 2 to 3min, 1 times / d, 10 times as a course of treatment.

**2. The wedge-shaped defect stannous fluoride treatment.** It is reported that a low concentration of stannous fluoride to effectively control dentin hypersensitivity. Sodium fluoride, stannous fluoride, sodium monofluorophosphate toothpaste teeth or partial erasure, some degree of desensitization.

**3. Calcium hydroxide treatment of wedge-shaped defects** can accelerate dentin mineralization, reduce the permeability of dentin, generally with distilled water and transferred into a paste of calcium hydroxide paste with a brush to brush is to dry the allergy tooth surface and maintain 5min, and then paste remove and rinse 1 a day, a week for a course of treatment. Also, it was argued, paste in root surface friction or used for periodontal surgery dressings will be a good tune to the prevention or treatment of dentine hypersensitivity.

**4. Strontium chloride treatment of wedge-shaped defects.** Has been confirmed that the strontium can penetrate calcified tissue (dentin). Strontium is deposited in a small tube, to reduce the permeability of the dentin. Also found that the application of the calcium and strontium, the mineralization to be higher than when using calcium alone, strontium ions in the accelerating calcification, or even block the dentinal tubules works.

**5. Resins and adhesives treatment of wedge-shaped defects.** Without filler tooth surface coating resin (such as epoxy resin), medical adhesive, etc., can be closed dentinal tubules, immediately effective, effective time up to one year, after falling reusable has been reported light-cured dentin bonding agent to bond the dentin surface coating light 20s, generally 1 or 2 can be effective.

**6. Iontophoresis treatment of wedge-shaped defects.** This method is more complex and needs a long time, apply to full mouth of teeth, or the majority of the teeth sensitive the general import fluoride or calcium, or both alternating import. Has been reported, and then import with 2% sodium fluoride ionizing light cured dentin bonding agent is applied to the light, the effect is very good.

When left untreated, the wedge-shaped defect continues to deepen to the point of absurd grooves that frequently provoke the formation of secondary dentin in the root canal. An overlay of caries accelerates the process. In most cases, the progress of the formation of the wedge-shaped defect cannot even be stopped with a correction of the tooth brushing method. The bristles of the tooth brush are pressed together in the already existing wedge-shaped indentation (like in a guiding groove) and the sawing and abrasive effects can hardly be avoided, even if techniques recommended for periodontal diseases are applied.

Besides endangering the pulp, a fracture of the tooth may also occur due to the wedge-shaped defect. Up to now, there is a widespread opinion

that only larger wedge-shaped defects must be treated with a filling or a crown.

After trials lasting several years, the experience gained in treating wedge-shaped defects with glass ionomer cements give rise to hope. These materials adhere to dentin and enamel, when they are freed from organic deposits by having been processed with a cleaning compound and being dry on the surface. Whether an additional pretreatment with citric acid improves the bond cannot yet be said with the necessary certainty.

### **Question 15. Teeth erosion.**

***K03.2 Erosion of teeth.*** Teeth erosion – loss of surface tooth structure by chemical action in the continued presence of demineralizing agents(acids). It is one of the most predominant oral pathologic changes. There is no convincing etiology, and multiple factors have been theorized for its pathogenesis.

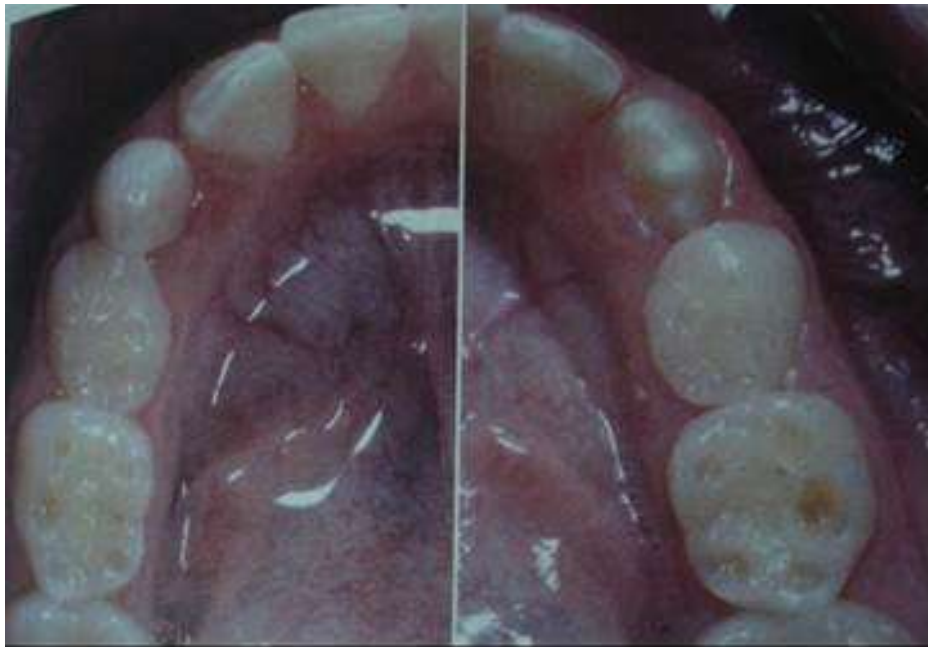
**Etiology.** Mechanical factors: the action of the muscles of lips and cheeks, and of tooth brush against affected surfaces.

**Chemical factors:**

- Ingested acids: citric acids (lemon and citrus fruits ) esp. If use in large amounts , can precipitate or initiate erosive lesion
- Secreted acids: the acidity of crevicular fluid has been correlated to cervical erosion.
- Acid fumes: acid vapours from nitric acid and sulphuric acids, acting in the mouths of workers in the factories ,where these acids are largely used or manufactured
- Refused acids: as a result of chronic , frequent regurgitation ,the stomach's hydrochloric acid can hit the teeth at specific locations ( atypical pattern of erosion affecting buccal surfaces of lower posterior teeth)
- The latter defective surfaces are associated with gastro esophageal reflux.



*Figure 86. Occlusal view of maxillary dentition exhibiting concave dentin depressions surrounded by elevated rims of enamel*



*Figure 87. Multiple cupped out depressions corresponding to the cusp tip.*



*Figure 88. Extensive loss of enamel and dentin on the buccal surface of maxillary bicuspid. (pt had sucked chronically on tamarinds).*



*Figure 89. Palatal surfaces of maxillary dentition in which the exposed dentin exhibits a concave surface and a peripheral white line of enamel.*

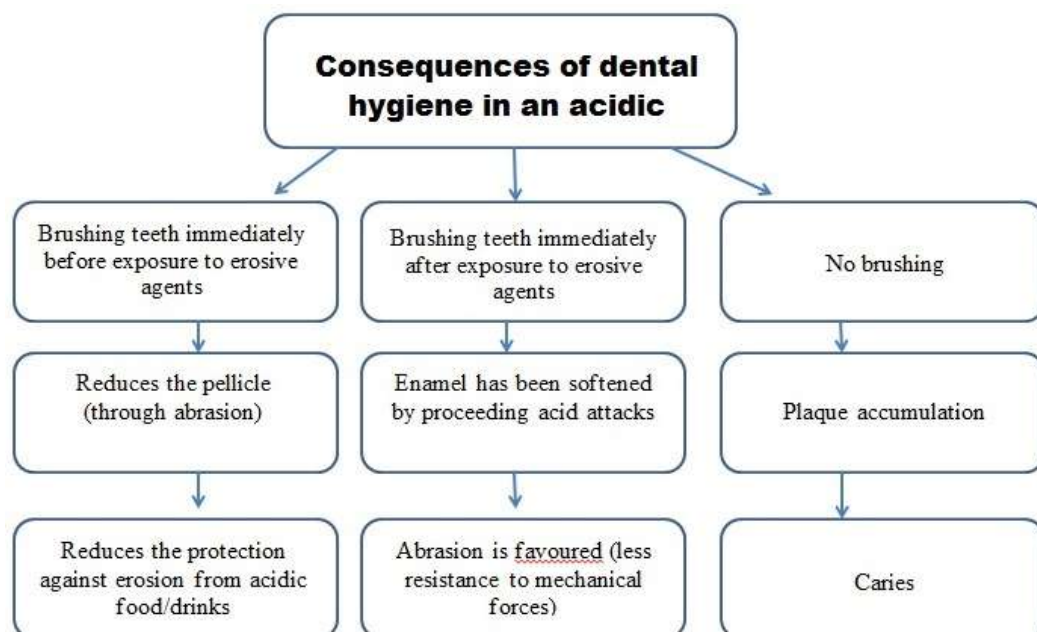
*The other substances that can corrode teeth.*

- chewable vitamin C tablets.
- aspirin tablets.
- aspirin powders.
- use of the amphetamine drug Ecstasy.

*Protocol for the prevention of progression of erosion*

- Diminish the frequency and severity of acid challenge.
  - Decreasing the amount and frequency of acidic foods or drinks.
  - Acidic drinks should be drunk quickly rather than sipped.
  - Using of straw reduces erosive potential.

- Treating the underlying medical disorder or disease.
- Enhance the defense mechanisms of body:
  - Saliva provides buffering capacity → increases with salivary flow rate.
  - Saliva supersaturated with Ca, P → inhibits demineralization of tooth structure.
  - Stimulation of salivary flow → sugarless lozenge, candy/gum is recommended
- Enhance acid resistance, remineralization and rehardening of the tooth surfaces.
  - Daily use topical fluoride at home
  - Fluoride application in office- 2-4 times a year, fluoride varnish recommended.
- Decrease abrasive forces.
  - Use a soft bristled toothbrush and brush gently.
  - No brushing should be done immediately after consuming acidic food and drink as teeth will be softened.
  - Rinsing with water is better than brushing after consuming acidic foods and drinks.



- Improve chemical protection
  - Neutralize acids in mouth - dissolving sugar free antacid tablets 5 times a day, particularly after an intrinsic or extrinsic acid challenge.
  - Dietary components- hard cheese (provides Ca and PO<sub>4</sub>), held in mouth after acidic challenge.
- Mechanical protection



- By application of composites and direct bonding where appropriate – to protect exposed dentin
- Occlusal guard /Acrylic splint in the form of stabilization splint necessary to protect dentition from further damage due to erosion.
- Monitor stability:
  - Use of casts/photos to document tooth wear status.
  - Regular recall examinations to review diet, oral hygiene methods, compliance with medications, topical flouride and splint usage.
- Restoration
  - Metallic restorations should be the choice of material, if restoration indicated (more resistant to erosion).
  - Tooth colored materials may also be used with minimal or no tooth preparation, with the assumption that restoration may require periodic replacement.

### **Question 16. Teeth abfraction**

Abfraction is a theoretical concept explaining a loss of tooth structure not caused by tooth decay (non-carious cervical lesions). It is suggested that these lesions are caused by forces placed on the teeth during biting, eating, chewing and grinding; the enamel, especially at the cementoenamel junction (CEJ), undergoes large amounts of stress, causing micro fractures and tooth tissue loss.



*Figure 90. Teeth abfraction.*

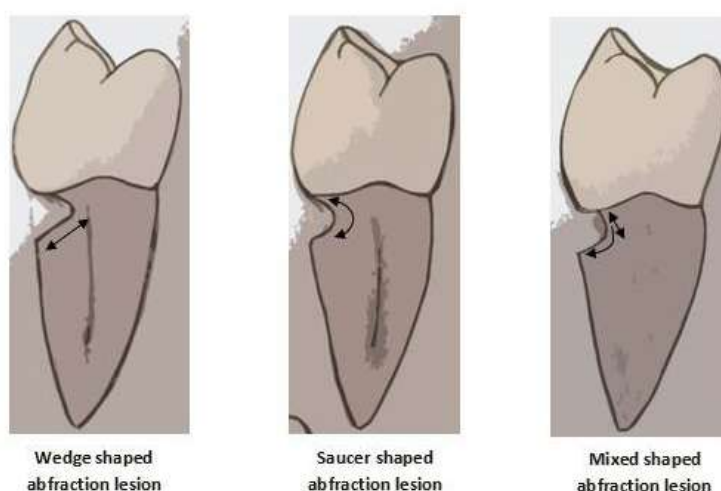
Abfraction is a form of non-carious tooth tissue loss that occurs along the gingival margin. In other words, abfraction is a loss of tooth structure that is not caused by tooth decay, located along the gum line. There has been a lot of theoretical evidence to support the concept of abfraction, but little experimental evidence exists.

The term abfraction was first published in 1991 in a journal article dedicated to distinguishing the lesion. The article was titled "Abfractions: A New Classification of Hard Tissue Lesions of Teeth" by John O. Grippo. This article introduced the definition of abfraction as a "pathologic loss of hard tissue tooth substance caused by bio mechanical loading forces". This



article was the first to establish abfraction as a new form of lesion, differing from abrasion, attrition, and erosion.

Tooth tissue is gradually weakened causing tissue loss through fracture and chipping or successively worn away leaving a non-carious lesion on the tooth surface. These lesions occur in both the dentine and enamel of the tooth. These lesions generally occur around the cervical areas of the dentition.



*Figure 91. Types of abfraction lesions.*

Abfraction lesions will generally occur in the region on the tooth where the greatest tensile stress is located. In statements such as these there is no comment on whether the lesions occur above or below the CEJ. One theory suggests that the abfraction lesions will only form above the CEJ. However, it is assumed that the abfraction lesions will occur anywhere in the cervical areas of affected teeth. It is important to note that studies supporting this configuration of abfraction lesions also state that when there is more than one abnormally large tensile stress on a tooth two or more abfraction lesions can result on the one surface.

When looking at abfraction lesions there are generally three shapes in which they appear, appearing as either wedge, saucer or mixed patterns. Wedge and saucer shaped lesions are the most common, whereas mixed lesions are less frequently identified in the oral cavity. Wedge shaped lesions have the sharpest internal line angles and saucer/mixed shaped lesions are either smooth internally, or a variety.

Clinically, people with abfraction lesions can also present with tooth sensitivity in the associated areas. This occurs because as the abfraction lesions appear, dentine/cementum is exposed. The dentine and cementum are less dense than tooth enamel and therefore more susceptible to sensation from thermal/mechanical sources.

**Causes.** As abfraction is still a controversial theory there are various ideas on what causes the lesions. Because of this controversy the true causes of abfraction also remain disputable. Researchers have proposed that abfraction is caused by forces on the tooth from the teeth touching together, occlusal forces, when chewing and swallowing. These lead to a concentration of stress and flexion at the area where the enamel and cementum meet (CEJ). This theoretical stress concentration and flexion over time causes the bonds in the enamel of the tooth to break down and either fracture or be worn away from other stressors such as erosion or abrasion. The people who initially proposed the theory of abfraction believe the occlusal forces alone cause the lesions without requiring the added abrasive components such as toothbrush and paste or erosion.

If teeth come together in a non-ideal bite the researchers state that this would create further stress in areas on the teeth. Teeth that come together too soon or come under more load than they are designed for could lead to abfraction lesions. The impacts of restorations on the chewing surfaces of the teeth being the incorrect height has also been raised as another factor adding to the stress at the CEJ.

Further research has shown that the normal occlusal forces from chewing and swallowing are not sufficient to cause the stress and flexion required to cause abfraction lesions. However, these studies have shown that the forces are sufficient in a person who grinds their teeth (bruxism). Several studies have suggested that it is more common among those who grind their teeth, as the forces are greater and of longer duration. Yet further studies have shown that these lesions do not always appear in people with bruxism and others without bruxism have these lesions.

There are other researchers who would state that occlusal forces have nothing to do with the lesions along the CEJ and that it is the result of abrasion from toothbrush with toothpaste that causes these lesions.

Being theoretical in nature there is more than one idea on how abfraction presents clinically in the mouth. One theory of its clinical features suggests that the lesions only form above the cemento-enamel junction (CEJ) (which is where the enamel and cementum meet on a tooth). If this is kept in mind, it serves as a platform for it to be distinguished from other non-caries lesions, such as tooth-brush abrasion.

**Treatment.** Treatment of abfraction lesions can be difficult due to the many possible causes. To provide the best treatment option the dental clinician must determine the level of activity and predict possible progression of the lesion.

It is usually recommended when an abfraction lesion is less than 1 millimeter, monitoring at regular intervals is a sufficient treatment option. If there are concerns around aesthetics or clinical consequences such as dentinal

hypersensitivity, a dental restoration (white filling) may be a suitable treatment option.

Aside from restoring the lesion, it is equally important to remove any other possible causative factors. Adjustments to the biting surfaces of the teeth alter the way the upper and lower teeth come together, this may assist by redirecting the occlusal load. The aim of this is to redirect the force of the load to the long axis of the tooth, therefore removing the stress on the lesion. This can also be achieved by altering the tooth surfaces such as cuspal inclines, reducing heavy contacts and removing premature contacts. If bruxism is deemed a contributing factor an occlusal splint can be an effective treatment for eliminating the irregular forces placed on the tooth.

### **Question 17. Pathological resorption of teeth.**

**K03.3 Pathological resorption of teeth.** Tooth resorption is a process by which all or part of a tooth structure is lost due to activation of the body's innate capacity to remove mineralized tissue, as mediated via cells such as osteoclasts.

Types include external resorption and internal resorption. It can be due to trauma, infection, or hyperplasia.



*Figure 92. The maxillary left lateral incisor (right in photograph) is afflicted with internal resorption*

**Internal resorption** is an unusual condition where the dentin and pulpal walls begin to resorb centrally within the root canal. The first evidence of the lesion may be the appearance of a pink-hued area on the crown of the tooth; the hyperplastic, vascular pulp tissue filling in the resorbed areas. This condition is referred to as a pink tooth of Mummery, after the 19th century anatomist James Howard Mummery.

The cause can sometimes be attributed to trauma to the tooth, but other times there is no known etiology. If the condition is discovered before perforation of the crown or root has occurred, endodontic therapy (root canal therapy) may be carried out with the expectation of a fairly high success rate.

The fact remains that for many afflicted by internal resorption, the cause is actually unknown as it cannot be tied to a specific injury or traumatic incident.

**External resorption** in dentistry, external resorption, or root resorption, is the breakdown or destruction and subsequent loss of the root structure of a tooth. This is caused by living body cells attacking part of the tooth. Severe root resorption is very difficult to treat and often requires the extraction of teeth.

Root resorption occurs as a result of differentiation of macrophages into (odontoclasts) in surrounding tissue which, if in close proximity to the root surface will resorb the root surface cementum and underlying root dentin. This can vary in severity from evidence of microscopic pits in the root surface to complete devastation of the root surface.

Deciduous root resorption is a natural process which allows exfoliation of the primary teeth to make way for the secondary teeth. It is caused by osteoclast differentiation due to pressure exerted by the erupting permanent tooth.

Root resorption of secondary teeth can occur as a result of pressure on the root surface. This can be from trauma, ectopic teeth erupting in the path of the root; chronic inflammation; excessive occlusal loading; trauma; improper reimplantation; aggressive tumors, cysts, and/or other growths; and/or unknown causes. The most common cause in Western society is orthodontic forces.

Roots of teeth are covered with cementum, a structure that resembles bone. However, cementum is more resistant to resorption than bone. There are a number of theories as to why this is the case. The most common hypothesis is that because cementum is harder and more mineralized than bone, and has anti-angiogenic properties, blood vessels are inhibited from forming adjacent to cementum, which in turn prevents access to osteoclasts.

## **Question 18. Hypercementosis.**

### ***K03.4 Hypercementosis***

Hypercementosis is an idiopathic, non-neoplastic condition characterized by the excessive buildup of normal cementum (calcified tissue) on the roots of one or more teeth. A thicker layer of cementum can give the tooth an enlarged appearance, which mainly occurs at the apex or apices of the tooth.

**Etiology.** Can be caused by many things.

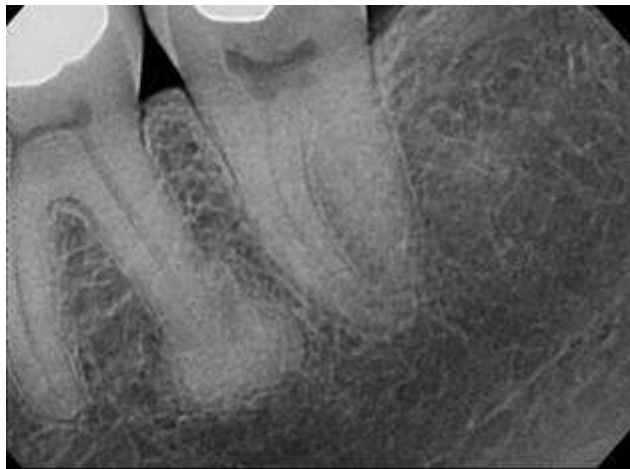
**Local factors:**

- Occlusal Trauma
- Trauma
- Non-functional tooth

- Unopposed tooth (and impacted teeth, embedded teeth, teeth without antagonists)

#### Systematic factors

- Idiopathic
- Pituitary Gigantism
- Paget's Disease
- Acromegaly
- Periapical granuloma
- Arthritis
- Calcinosis
- Rheumatic fever



*Figure 92. Hypercementosis.*

It may be one of the complications of Paget's disease of bone in the form of generalized hypercementosis. It may also be a compensatory mechanism in response to attrition to increase occlusal tooth height.

**Symptoms.** It is experienced as an uncomfortable sensation in the tooth, followed by an aching pain. It may be noted on radiographs as a radiopaque (or lighter) mass at each root apex.

**Complications.** Such deposits form bulbous enlargements on the roots and may interfere with extractions, especially if adjacent teeth become fused (concrecence). It may also result in pulpal necrosis by blocking blood supply via the apical foramen.

### **Question19. Ankylosis of teeth.**

***K03.5 Ankylosis of teeth.*** Tooth ankylosis refers to a fusion (ankylosis) of teeth to bone. The condition is diagnosed with radiographs (X-rays), which show loss of the periodontal ligament space and blending of the root with the bone. Clinically the tooth sounds solid when percussed (tapped) compared to

the dull, cushioned sound from normal teeth. Ankylosis of teeth is uncommon, more so in deciduous teeth than permanent teeth.

***Deciduous (baby) teeth.*** Ankylosis of deciduous teeth ("submerged teeth") may rarely occur. The most commonly affected tooth is the mandibular (lower) second deciduous molar. Partial root resorption first occurs and then the tooth fuses to the bone. This prevents normal exfoliation of the deciduous tooth and typically causes impaction of the permanent successor tooth. As growth of the alveolar bone continues and the adjacent permanent teeth erupt, the ankylosed deciduous tooth appears to submerge into the bone, although in reality it has not changed position. Treatment is by extraction of the involved tooth, to prevent malocclusion, periodontal disturbance or dental caries.



*Figure 93. Ankylosis of deciduous teeth.*

***Permanent (adult) teeth.*** Repair with cementum or dentin occurs after partial root resorption, fusing the tooth with the bone. It may occur following dental trauma, especially occlusal trauma, or after periapical periodontitis caused by pulp necrosis. Ankylosis itself is not a reason to remove a permanent tooth, however teeth which must be removed for other reasons are made significantly more difficult to remove if they are ankylosed.

### **Question20. Trauma of teeth.**

Trauma to the oral region occurs frequently and comprises 5% of all injuries for which people seek treatment. In preschool children the figure is as high as 18% of all injuries. Amongst all facial injuries, dental injuries are the most common of which crown fractures and luxations occur most frequently. An appropriate treatment plan after an injury is important for a good prognosis. Guidelines are useful for dentists and other health care professionals in delivering the best care possible in an efficient manner. The International Association of Dental Traumatology (IADT) has developed a

consensus statement after a review of the dental literature and group discussions. The first set of guidelines was published by IADT in 2001. Experienced researchers and clinicians from various specialties were included in the group. In cases where the data did not appear conclusive, recommendations were based on the consensus opinion of the IADT board members. The guidelines represent the current best evidence, based on literature research and professional opinion. As is true for all guidelines, the health care provider must apply clinical judgment dictated by the conditions present in the given traumatic situation. The IADT does not guarantee favorable outcomes from following the Guidelines, but using the recommended procedures can maximize the chances of success. Because management of permanent and primary dentition differs significantly.

***Uncomplicated crown fracture.*** Fracture involves enamel or dentin and enamel; the pulp is not exposed. Sensibility testing may be negative initially indicating transient pulpal damage; monitor pulpal response until a definitive pulpal diagnosis can be made.

***Treatment:*** If tooth fragment is available, it can be bonded to the tooth. Urgent care option is to cover the exposed dentin with a material such as glass ionomer or a permanent restoration using a bonding agent and composite resin. Definitive treatment for the fractured crown may be restoration with accepted dental restorative materials

***Complicated crown fracture.*** Fracture involves enamel and dentin and the pulp is exposed. Sensibility testing is usually not indicated initially since vitality of the pulp can be visualized. Follow up control visits after initial treatment includes sensibility testing to monitor pulpal status.

***Treatment:*** In young patients with immature, still developing teeth, it is advantageous to preserve pulp vitality by pulp capping or partial pulpotomy. This treatment is also the choice in young patients with completely formed teeth. Calcium hydroxide and MTA (white) are suitable materials for such procedures.

In older patients, root canal treatment can be the treatment of choice, although pulp capping or partial pulpotomy may also be selected. If too much time elapses between accident and treatment and the pulp becomes necrotic, root canal treatment is indicated to preserve the tooth. In extensive crown fractures a decision must be made whether treatment other than extraction is feasible.

***Crown-root fracture.*** Fracture involves enamel, dentin and root structure; the pulp may or may not be exposed. Additional findings may include loose, but still attached, segments of the tooth. Sensibility testing is usually positive.

***Treatment:*** Treatment recommendations are the same as for complicated crown fractures (see above). In addition, attempts at stabilizing

loose segments of the tooth by bonding may be advantageous, at least as a temporary measure, until a definitive treatment plan can be formulated

**Root fracture.** The coronal segment may be mobile and may be displaced. The tooth may be tender to percussion. Sensibility testing may give negative results initially, indicating transient or permanent pulpal damage; monitoring the status of the pulp is recommended. Transient crown discoloration (red or grey) may occur

**Treatment:** Reposition, if displaced, the coronal segment of the tooth as soon as possible. Check position radio graphically. Stabilize the tooth with a flexible splint for 4 weeks. If the root fracture is near the cervical area of the tooth, stabilization is beneficial for a longer period of time (up to 4 months).

It is advisable to monitor healing for at least 1 year to determine pulpal status. If pulp necrosis develops, root canal treatment of the coronal tooth segment to the fracture line is indicated to preserve the tooth.

### **Tests to the topic**

#### **1. The main cause of fluorosis is:**

- a. Excessive fluorine consumption.
- b. Poor oral hygiene.
- c. Insufficient fluorine consumption.

#### **2. What types of enamel hypoplasia according to the etiology do you know?**

- a. Systemic.
- b. Localized.
- c. General.
- d. All answers are right.

#### **3. Grading of mottled enamel is**

- a. Very mild
- b. Mild
- c. Moderate
- d. Severe
- e. All answers are right

#### **4. According to the dean's index mild fluorosis is characterized by:**

- a. All tooth surfaces affected; marked wear on biting surfaces; brown stain may be present.
- b. Small opaque, paper white areas covering less than 25% of the tooth surface.
- c. Opaque white areas covering less than 50% of the tooth surface.



**5. What teeth are the most susceptible to the hypoplasia?**

- a. The first permanent molars.
- b. Upper central incisors.
- c. Second molars.
- d. Lower lateral incisors and canines.
- e. All answers are right.

**6. What types of filling materials are used for treatment of abrasion?**

- a. Microfilled composites.
- b. Flowable resins.
- c. Glass ionomer cements.
- d. All answers are right.

**7. What is the main cause of erosion?**

- a. Acids.
- b. Fluorides.
- c. Consumption of tetracycline.
- d. All answers are right.

**8. What is the main cause of abfraction?**

- a. Unproper tooth brushing.
- b. Acids.
- c. Streptococcus mutans.
- d. Excessive vertical load on tooth.
- e. All answers are right.

**9. Internal resorption is a condition which characterized:**

- a. The dentin and pulpal walls begin to resorb centrally within the root canal.
- b. The breakdown or destruction and subsequent loss of the root structure of a tooth.
- c. All answers are right.

**10. External resorption is a condition which characterized:**

- a. The dentin and pulpal walls begin to resorb centrally within the root canal.
- b. The breakdown or destruction and subsequent loss of the root structure of a tooth.
- c. All answers are right.

## Test answers

Questions									
1	2	3	4	5	6	7	8	9	10
<b>Lesson 1</b>									
b	b	a	a	b	b	c	b	d,e	e
<b>Lesson 2</b>									
a,c,d	c,d	g	a,b,d	c	c	d	d	a,b	g
<b>Lesson 3</b>									
g	a-d	a,b,d	c	h	d	c	g	e	d
<b>Lesson 4</b>									
a	c	a	d	a	b	c	a	a	e
<b>Lesson 5</b>									
a,d	a,d	a	e	b	a-c	a-c	a,c	a	a
<b>Lesson 6</b>									
b	d	b	c	b	c	d	a	b	a
<b>Lesson 7</b>									
d	f	g	f	e	h	a,c	d	e	c
<b>Lesson 8</b>									
a-d	d	c	b	c	d	d	b	b,d	a
<b>Lesson 9</b>									
c	a	e	d	e	a	d	a	a,b	a
<b>Lesson 10</b>									
c	a	a	a	a,b	a,b	a	a	a	e
<b>Lesson 11</b>									
a-c	a	e	d	e	e	a-b	a-c	a,c	a,b
<b>Lesson 12</b>									
a,c,d	a,b,d,e	a,c,d	a-f	a-f	b-d	a,b,e	a-f	d	d
<b>Lesson 13</b>									
b	d	a,c	d	f	a	e	a	a	a
<b>Lesson 14</b>									
c	d	b	f	e	b	d	e	a	b-e
<b>Lesson 15</b>									
e	e	g	d	d	d	b	d	d	c
<b>Lesson 16</b>									
a	b	e	e	e	a	d	a	c	b
<b>Lesson 17</b>									
d	a,b	d	j	f	a,b,d,e	e	e	e	e
<b>Lesson 18</b>									
d	d	a	a,b	b-d	a	a	a	d	a-c
<b>Lesson 19</b>									
a	d	e	c	a,b,d	d	a	d	a	b

## References

1. Апикальный периодонтит: этиология, патогенез, классификация = Apical periodontitis: etiology, pathogenesis, classification: учеб.–метод. пособие / Л.А. Казеко [и др.]; Белорус. гос. мед. ун–т, 1–я каф. терапевт. стоматологии. – 2–е изд. – Минск: БГМУ, 2016. – 15 с.
2. Бондарик, Е.А. Болезни зубов некариозного происхождения: учеб.–метод. пособие / Е. А. Бондарик, Е. В. Шумакова, А. Г.Третьякович; Белорус. гос. мед. ун–т, 2–я каф. терапевт. стоматологии. – Минск: БГМУ, 2010. – 48 с.
3. Дедова Л.Н. Кариес корня: клиника, диагностика, лечение: учеб.–метод. пособие / Л.Н. Дедова, О. В. Кандрукевич; Белорус. гос. мед. ун–т, 3–я каф. терапевт. стоматологии. – Минск: БГМУ, 2013. – 39 с.
4. Казеко, Л.А. Апикальный периодонтит: диагностика, клинические проявления, лечение = Apical periodontitis: diagnostics, clinical manifestations, treatment: учеб.–метод. пособие / Л.А. Казеко, Ю.В. Модринская, К.В. Севрукевич; Белорус. гос. мед. ун–т, 1–я каф. терапевт. стоматологии. – Минск: БГМУ, 2015. – 15 с.
5. Казеко, Л.А. Кариес зубов: клиника, диагностика, прогнозирование, лечение = Dental caries: clinical picture, diagnosis, prediction, treatment: учеб.–метод. пособие / Л.А. Казеко, Ю.В. Модринская, К.В. Севрукевич; Белорус. гос. мед. ун–т, 1–я каф. терапевт. стоматологии. – Минск: БГМУ, 2014. – 29 с.
6. Казеко, Л.А. Кариес: этиология, патогенез, профилактика = Dental caries: etiology, pathogenesis, prevention: учеб.–метод. пособие / Л. А. Казеко, К. В. Севрукевич ; Белорус. гос. мед. ун–т, 1–я каф. терапевт. стоматологии. – Минск: БГМУ, 2014. – 19 с.
7. Казеко, Л.А. Методики работы с амальгамой в терапевтической стоматологии: учеб.-метод. пособие / Л.А. Казеко, С.Н. Храмченко. – Минск: БГМУ, 2011. – 27 с.
8. Казеко, Л.А. Профессиональная гигиена = Professional oral hygiene: учеб.-метод. пособие / Л.А. Казеко, О.А. Тарасенко. – Минск: БГМУ, 2016. – 42 с.
9. Казеко, Л.А. Пульпит: этиология, патогенез, классификация = Pulpitis: etiology, pathogenesis, classification: учеб.–метод. пособие / Л.А. Казеко, Ю.В. Модринская, К.В. Севрукевич, Белорус. гос. мед. ун–т, 1–я каф. терапевт. стоматологии. – Минск: БГМУ, 2014. – 18 с.
10. Князева, М.А. Алгоритм описания рентгенограмм в клинике терапевтической стоматологии. Учебно–методическое пособие для

- студентов стоматологического факультета (часть I) / М.А.Князева, Ю.П.Чернявский. – Витебск: ВГМУ, 2011. – 58 с.
11. Колб, Е.Л. Амальгама в терапевтической стоматологии = Amalgam in clinical dentistry: учебно-методическое пособие / Е. Л. Колб, Т. И. Гунько, И. С. Кармалькова. – 2-е изд. – Минск: БГМУ, 2017. – 28 с.
  12. Николаев, А.И. Практическая терапевтическая стоматология: учеб. пособие / А.И. Николаев, Л.М. Цепов. – 9-е изд. – М.: МЕДпресс-информ, 2014. – 928 с.
  13. Основы профессиональной гигиены полости рта = BasicsofProfessionalOralHygiene: учебно-методическое пособие / С. С. Лобко [и др.]. – Минск: БГМУ, 2019. – 31 с.
  14. Терапевтическая стоматология. Учебник для медицинских вузов / под редакцией Е. В. Боровского – М.: ООО «Медицинское информационное агентство», 2011. – 840 с.
  15. Чернявский, Ю.П. Асептика и антисептика в терапевтической стоматологии: пособие / Ю.П. Чернявский, Т.И. Першукевич. – Витебск: ВГМУ, 2014. – 194 с.
  16. Чернявский, Ю.П. Курс лекций по терапевтической стоматологии: Пособие. Часть 1 / Ю.П. Чернявский. – Витебск: ВГМУ, 2013. – 377 с.
  17. Чернявский, Ю.П. Курс лекций по терапевтической стоматологии: Пособие. Часть 2 / Ю.П. Чернявский. – Витебск: ВГМУ, 2013. – 194 с.
  18. Garg, Nisha. Textbook of Endodontics / Nisha Garg, Amit Garg. – Jaypee Brothers Medical Publishers, 2010. – 540 p.
  19. Gufaran Ali, Syed. Pulpitis: A review / Syed Gufaran Ali, Sanjyot Mulay // Journal of Dental and Medical Sciences. – 2015. - Volume 14, Issue 8 Ver. VI . – P. 92-97.
  20. Hargreaves, K.M. Cohen's pathways of the pulp / K.M. Hargreaves, L.H. Berman, I. Rotstein. – 11th Edition. – Elsevier, 2016. – 1143 p.
  21. Heymann, H.O. Sturdevant's art and science of operative dentistry / H.O. Heymann. – Mosby, 2013. – P. 216-254, 339-455.
  22. Marya, C.M. Public Health Dentistry / C.M. Marya. – India: Jaypee Brothers Medical Publishers, 2012. – 248 p.
  23. Shantipriya, Reddy. Essentials of Clinical Periodontology and Periodontics / Reddy Shantipriya. –Jaypee Brothers Medical Publishers (P) Ltd, 2011. – 514 p.
  24. Volkova, M.N. Guideline on therapeutic dentistry for the 5-th term: educational and methodical edition / M.N. Volkova, N.A. Sakharuk, N.A. Korenevskaya. – Vitebsk: VGMU, 2016. – 250 с.

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**ТЕРАПЕВТИЧЕСКАЯ СТОМАТОЛОГИЯ  
ДЛЯ СТУДЕНТОВ 3 КУРСА**

**THERAPEUTIC DENTISTRY FOR THE 3RD YEAR STUDENTS**

**Учебно-методическое пособие на английском языке**

**Часть 1**

Редактор М.Н. Волкова  
Компьютерная верстка С.В. Суслина

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